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STRESS IN MAMMALS: THE POTENTIAL INFLUENCE OF FISHERY-INDUCED STRESS ON DOLPHINS IN THE EASTERN TROPICAL PACIFIC OCEAN

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U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
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ABSTRACT

There is concern that fishing methods used by the eastern tropical Pacific Ocean (ETP) tuna purse-seine fishery may cause stress to dolphins, and that such stress may be having an adverse impact on reproduction or survival in these mammals. Recent legislation, the 1997 International Dolphin Conservation Program Act, required this review of stress-related research to provide a context for future scientific findings. This review includes background information on the ETP tuna fishery and the dolphins involved. General information regarding stress theory and the physiology of stress is also included. Four general areas of study are reviewed that provide information on physiological and behavioral responses to stress that may be relevant to dolphins involved in the ETP tuna purse-seine fishery. These include laboratory research, research on domestic animals, clinical studies of stress effects in humans, and research on free-ranging mammal populations.

Potential stress effects of specific fisheries operations (search, chase, and capture) on the dolphins involved in the ETP fishery are considered. Search operations may cause disruption of habitat utilization, foraging activities, and social activities. Chase and capture operations may cause immediate or short-term physiological responses such as activation of the hypothalamic-pituitary-adrenal axis in response to psychological or social stressors. Psychosocial stressors include separation of mother and young, separation from social groups, social aggression during net confinement, and novelty. Other potential short-term responses of dolphins to chase and capture include severe muscle damage resulting in a condition known as capture myopathy and hyperthermia.

The potential sublethal effects of long-term stress include stress-induced pathologies, compromise to the immune system, as well as impaired reproduction, growth, and metabolism. Based on information from other mammals it is plausible that reproduction for some proportion of female dolphins will be disrupted, either as a result of the hypothalamic-pituitary-adrenal response to stress or through the development of pathologies resulting from chronic stress. Cow-calf separation can occur as the result of chase and capture, and it appears that young animals may be particularly vulnerable to impacts of fisheries operations. Maternal separation and novelty may induce significant hypothalamic-pituitary-adrenal responses in young animals, and this can result in impaired growth.

Although this review of existing literature regarding stress in mammals can not provide a quantitative answer to the question of whether the tuna fishery is causing stress to affected dolphin populations, the available information and evidence point to the likelihood that physiological stress is induced by fisheries activities. It is therefore plausible, that stress resulting from chase and capture in the ETP yellowfin tuna purse-seine fishery could have a population level effect on one or more dolphin stocks.

I. BACKGROUND INFORMATION

A. INTRODUCTION

In an effort to reduce dolphin mortality in the eastern tropical Pacific Ocean (ETP) tuna purse-seine fishery, an agreement called the Declaration of Panama was negotiated by the United States and eleven other fishing nations in 1995. The agreement imposes a total mortality limit of 5,000 dolphins per year and intends that all countries will take steps to eliminate mortality entirely. The International Dolphin Conservation Program Act (IDCP Act; U.S. Public Law 105-42), a 1997 amendment to the Marine Mammal Protection Act, was created to give effect to the Declaration of Panama by allowing the importation of currently embargoed yellowfin tuna into the United States. The law also includes provisions that could allow tuna caught by the intentional encirclement of dolphins with a purse-seine net in the ETP to be declared "dolphin safe" if no dolphins are observed to be killed or seriously injured in that set.

There is concern that fishing methods used by the ETP tuna-dolphin purse-seine fishery may cause stress to dolphins and that such stress may be having an adverse impact on reproduction or survival of these animals. As a result, the IDCP Act requires that research consisting of population abundance surveys (see Gerrodette *et al.*, 1998) and several "stress studies" be conducted by the National Marine Fisheries Service (NMFS) to determine whether the "intentional deployment on, or encirclement of, dolphins by purse-seine nets is having a significant adverse impact on any depleted dolphin stock." Initial findings, based on preliminary results of the research program, are to be made by March 31, 1999, with final conclusions and recommendations reported to Congress by December 31, 2002.

Among the stress studies required by the IDCP Act is a review of relevant stress-related research. This report addresses that requirement by reviewing current knowledge of stress in mammals, focusing on scientific information regarding physiological responses to stress and the potential consequences of fishery-induced stress to dolphins involved in the ETP tuna-purse-seine fishery. The overall objective of this review is to provide a context for future scientific findings by describing what is known about physiological and behavioral responses to stress in mammals and relating that information to the chase and encirclement of dolphins in the ETP fishery.

Investigations of stress have been numerous and diverse, ranging from controlled laboratory studies of neuroendocrine processes to field observations of behavior. The multitude of work on the subject precludes a comprehensive review of research on stress in mammals. Rather, the intention here is to provide background information and to document relevant examples of scientific evidence that pertain to the potential for fishery-induced stress and resultant physiological responses of affected dolphins in the ETP.

Section I of this review provides background information on the ETP tuna fishery and the dolphin stocks involved, and then summarizes the fisheries procedures related to chase and capture. As a basis for later discussion of the potential consequences of stress to dolphins involved in the fishery, this background section also includes a brief description of the concept of stress and the development of stress theory. The section outlines the four general areas of study that have provided primary information on the kinds of physiological or behavioral responses to stress that may be relevant to dolphins involved in the ETP tuna purse-seine fishery. These areas of study include laboratory

research, studies conducted to investigate the effects of stress on domestic animals, clinical studies of stress effects in humans, and research on free-ranging mammals. Lastly, the background section includes general information on the current understanding of stress physiology.

Section II proceeds to a discussion of stress responses that might be expected in ETP dolphins affected by the tuna purse-seine fishery and considers effects due to search, chase, and capture operations. Short-term (immediate) physiological responses are considered first (e.g. capture myopathy, thermoregulation), followed by potential long-term effects (e.g. stress-induced pathologies, changes in immune and reproductive function, and effects on growth).

Section III concludes the review with a general summary discussing the likely effects of fisheries-induced stress on individual dolphins in the ETP and potential effects on dolphin populations.

B. THE ETP YELLOWFIN TUNA FISHERY

A brief description of dolphin involvement in the ETP yellowfin tuna purse-seine fishery and a general outline of fisheries operations are provided here, as they are useful for examining aspects of the fishery that could cause stress to dolphins (Table 1). Joseph (1994) and Gosliner (in press) have provided descriptions of the recent history of conservation issues surrounding the fishery and several additional references describe the fishery and fisheries operations in varying detail (Coe *et al.*, 1984; National Research Council, 1992; Ben-Yami, 1994; Hall; 1998).

1. Dolphins and The ETP Tuna Purse-Seine Fishery

Because there is an association between tuna and dolphins, fishermen in the tuna industry often set purse-seine nets on dolphin schools as a means of catching tuna (Perrin, 1969). Pantropical spotted, *Stenella attenuata*, spinner, *S. longirostris*, and common, *Delphinus* spp., dolphins are the species most often found in association with tuna (predominately yellowfin, *Thunnus albacares*; Hammond, 1981; Au and Pitman, 1988). The reason for the bond and the relative importance of it for either dolphins or tuna is not known (Hammond, 1981; but see Au and Pitman, 1986; Au 1991; Edwards, 1992).

Perrin *et al.* (1985), Perrin *et al.* (1991), and Dizon *et al.* (1994) have described the stocks involved in the ETP tuna fishery. Three stocks are considered to be depleted: the northeastern offshore spotted dolphin, and the eastern and coastal spinner dolphin stocks. Wade and Gerrodette (1993) estimated 730,000 northeastern offshore spotted dolphins and 630,000 eastern spinner dolphins in the ETP. Current estimates are not established (but see Gerrodette *et al.,* 1998). The northeastern spotted dolphin is the target of approximately 85% of all sets on dolphins. Spotted dolphins occur mostly in single-species herds, but a significant fraction of schools also include eastern spinner dolphins.

Historically, levels of dolphin mortality in purse-seine nets were extremely high (Smith, 1983; Lo and Smith, 1986; Wade, 1995). Wade (1995) estimated that 4.9 million dolphins were killed by the purse-seine fishery over a fourteen year period (1959-1972). Mortality decreased gradually because of measures such as the Marine Mammal Protection Act (1972) and use of equipment designed to prevent dolphin entanglement in purse-seine nets (Barham *et al.*, 1977). Despite these changes, dolphin mortality

continued to occur at levels that were probably higher than the stocks impacted by the fishery could sustain (Wade, 1995).

Since 1992, dolphin mortality in the fishery has decreased significantly to a few thousand animals per year (Hall and Lennert, 1993, 1994, 1997; Lennert and Hall, 1995, 1996). A number of legislative measures, including US embargoes and the advent of the "dolphin-safe" policy, caused changes in the fishery and mortality was greatly decreased (see Joseph, 1994). Wade (1995) concluded that these low levels of mortality could allow recovery and growth of the impacted stocks. Total estimated mortality for 1996 was 2,547 dolphins (Lennert and Hall, 1997) and the preliminary estimate for 1997 is approximately 3,000 dolphins (Inter-American Tropical Tuna Association, IATTC, 1997).

2. Fisheries Operations

a. *Search Procedures*

While searching for tuna from a purse-seiner, fishermen use binoculars and radar to detect cues. A helicopter is also often used to search for dolphin herds and to assess whether or not a herd carries sufficient tuna to make a set. When a herd associated with commercial quantities of tuna is located, the seiner approaches traveling at approximately 15 knots. Dolphins are reported to react to the vessels from a distance of approximately five to seven kilometers (Norris *et al.*, 1978).

b. *Chase and Capture Procedures*

Four to six speedboats (85-140 hp) are used to chase the dolphins, and to separate and direct the movements of the portion of the herd to be encircled. The helicopter may also be used to herd animals during these operations, which usually lasts from 20-40 minutes but can sometimes take more than two hours.

The purse-seine net is set with the aid of a skiff deployed from the net stack at the rear of the vessel. The net is approximately 1.6 km long by 200 m deep. The skiff acts as an anchor for one end of the net as the seiner moves away in a circle, setting the net continuously behind it. Speedboats herd the dolphins into the closing arc of the purse-seine (dolphins sometimes escape by running across the narrowing opening as the net is pulled into a circle and by diving under the net before it is pursed). Sometimes the entire herd is encircled or "wrapped" while other times only a portion of the herd is encircled. Dolphins tend to gather together in the net as it is pursed at the bottom (Pryor and Kang Shallenberger, 1991, observed that dolphins tended to gather at the surface and as far away from the vessel as was possible). Backdown, a procedure for releasing dolphins over the net's corkline, begins after about two-thirds of the net has been hauled aboard. A channel is formed at the far side of the net and the corkline is submerged so that the dolphins can exit. Speedboats may be used to pull the corkline, thus keeping the channel from collapsing. Crewmen may enter the water, when necessary, to pull dolphins over the corkline. Encirclement takes approximately 40 minutes and dolphins may be confined for about an additional hour (longer if unanticipated problems occur).

c. *Behavioral Observations of Dolphins During Capture*

Pryor and Kang Shallenberger (1991) observed the behavior of spotted dolphins in tuna purse-seine nets and reported (as did Norris *et al.*, 1978) that, when set on, all dolphins positioned themselves as far from the ship (the "alarming stimulus") as was possible. Signs of agitation on the part of the dolphins varied among sets and also among individuals. At the beginning of nine out of 17 observed sets, Pryor and Kang Shallenberger (1991) observed headslaps, tailslaps, thrashing, and bunching. The authors noted that these behaviors are considered to be signs of agitation, stress, and fear in captive *Stenella* spp. (see Norris and Dohl, 1980a).

Dolphins have been observed being hyperactive as well as passive when confined in purse-seine nets, and their appear to be some differences in behavior between spotted and spinner dolphins. Spinner dolphins have been observed to continue swimming and diving in the net, often with high activity levels (Norris *et al.,* 1978). Several passive behaviors (sleeping, sinking, and rafting) have been described, mostly for spotted dolphins encircled in purse-seine nets and it has been suggested that such behaviors may have been the product of stress (Norris *et al.*, 1978; Coe and Stuntz, 1980). There is anecdotal information that "sleeping" behavior, on the net bottom, has become rare. Rafting behavior is still often observed, and has been suggested to be a learned or conditioned behavior for avoiding net-entanglement, but has also been suggested to result from fisheries-induced stress (Coe and Stuntz, 1980; Sevenbergen, 1997). The significance and current prevalence of these behaviors is not known.

d. *Capture Rate*

There is very little information available to determine the capture rate of dolphin herds or individual dolphins in the ETP tuna purse-seine fishery. Dividing the number of dolphins in the ETP by the number of sets annually gives a simplistic capture rate of eight captures/animal/year. Recently, Perkins and Edwards (1997) estimated capture based on an analysis considering the fact that tuna fishermen actively search for, and set on, larger schools (and based on the assumption that individual dolphins choose to remain in schools of a particular size). Their analysis indicated that approximately 10% of the northeastern spotted dolphin stock may be set on as often as once per week (Perkins and Edwards, 1997). Approximately 30% of the stock may be set on 2-8 times per year, and about 50% of the stock may be set on once or twice a year. The analysis, however, is complicated by the fact that dolphin herds are not stable entities and may fluctuate in size as often as on a daily basis.

C. STRESS IN MAMMALS

The term "stress" is often used ambiguously to describe a broad range of conditions that threaten the well being of an organism. The lack of a cohesive definition of stress has led authors to caution that the word can be misleading or useless (e.g. Harvey *et al.* (1984). To avoid misinterpretation, researchers investigating physiological and behavioral responses to stress often have to precede descriptions of their investigations with an operational definition of stress, and there are published papers devoted to developing a unified definition of stress (Levine, 1985; Moberg, 1987a; Levine and Ursin, 1991).

Johnson *et al.* (1992) attributed these conceptual problems in stress research to confusion surrounding the definitions of "stress," "stressor," "adaptive responses," and the "consequences of stress." To clarify these definitions, Chrousos and Gold (1992) drew upon an ancient Greek concept, that "balance or harmony" is necessary for the survival of an organism, and defined stress as "a state of disharmony or threatened homeostasis," in their review of the concepts of stress and stress system dysregulation. Homeostasis is simply the maintenance or regulation of the body's set points. Following their recommendation, stress is defined in this review as a state of threatened homeostasis.

A stressor can be defined as any perturbation that disrupts homeostasis. Responses to stressors include a number of mechanisms by which the organism can regain homeostasis. The physiological systems that maintain homeostasis, "allostatic" systems, are activated when needed to regulate functions such as blood glucose levels, pH levels, and temperature. Hence, an organism's responses to stress are adaptive (acting to regain homeostasis) and can be both physiological and behavioral. The consequences of stress can be adverse, however, when the organism is unable to successfully regulate the stress response. This can occur when the allostatic systems become activated too frequently or remain active for too long as the result of stress is prolonged or dysfunction of the stress response.

1. Stressors

Not all stressors elicit the same physiological or behavioral responses. There are different types and degrees of stress, and these factors can influence an individual's

response to stress. Acute stress is sudden, whereas chronic stress is marked by long duration or frequent occurrence. An acute stressor may elicit an adaptive physiological response, but chronic stress may be pathogenic. The effects of both can be cumulative over time (Chrousos and Gold, 1992).

The type, intensity and duration of a stressor are important determinants of an individual's response to it. In addition, the interstressor interval is an important factor in determining the response to stress and has been demonstrated to be a significant factor in an individual's ability to become habituated to a stressor (De Boer *et al.*, 1990). In laboratory rodents, for example, exposure to chronic intermittent stressors can result in elevated levels of stress related hormones, or, via what is apparently habituation, in decreased or unchanged levels of these substances (see De Boer *et al.*, 1990).

2. Individual Temperament and Stress

Individuals differ in their responses to stressors (Hinkle, 1974), and this is partially related to individual variation in physical condition, physiology, and prior exposure to the stressor. But individual differences in response to stress are also influenced by a combination of physiological, sociological, behavioral, and environmental parameters (see Lyons *et al.*, 1988a,b; Hinkle, 1974). The psychological component of stress is important in this respect, because individual differences reflect differences in perception, or fear, of a stressor (Gray, 1982).

The work of J. W. Mason and other researchers in the early part of this century (see Mason, 1968a for review) illustrated the influence of psychological factors upon an individual's physiological response to stress. Mason (1968a) included novel experiences

and social factors, such as crowding or aggression, as potent psychological stimuli that have been demonstrated to elicit a physiological stress response in animals and humans. The perception of control or predictability over a situation can be important in this regard as well (see Mason, 1968a; Friend, 1991; Sapolsky, 1987a).

3. Stress Theory

a. *Previous Research*

Many authors have summarized the history of stress theory (see Moberg, 1985, Chrousos and Gold, 1992; Chrousos *et al.*, 1988; Johnson *et al.*, 1992) and it will only be addressed briefly here to provide context for the subsequent discussion of physiological stress and its consequences. Cannon (see Cannon and De La Paz, 1911) is often attributed with the early recognition of the connection between activity of the adrenal gland and stressors. He identified the connection between the sympathetic nervous system and the adrenal glands for organizing the "fight or flight" response to threat. It was Cannon who used the term "homeostasis" and demonstrated that both physical and emotional stimuli can elicit the same response.

Selye (1936) developed a stress theory, the General Adaptation Syndrome, that was based upon adrenal gland function in response to stimuli. The syndrome was defined as having three phases: 1) alarm reaction, characterized by an initial secretion of glucocorticosteroids (stress related hormones) from the adrenal gland, followed by a depletion of glucocorticosteroid reserves, 2) resistance, an adaptation phase during which the adrenal cortex could secrete increased levels of corticosteroids, and 3) adrenal exhaustion, at which point glucocorticoids were depleted. Selye used the terms

"eustress" and "distress," differentiating between harmless challenges or severe threats to homeostasis. Selye (1936, 1950, 1973) emphasized that stress is a nonspecific phenomenon because he considered that a wide variety of stimuli or stressors could initiate the stress syndrome.

Later, Mason (Mason 1968b, 1975) demonstrated that responses to stressors involve the entire neuroendocrine system and are variable, complex and integrated reactions that differ according to the type of stressor. The significant effect of emotional influences on pituitary-adrenal activity was illustrated in much of Mason's work (Mason, 1968a&b, 1975), which underscored the importance of psychological stressors (especially those that are novel, uncertain, and unpredictable) in eliciting a broad number of concurrent neuroendocrine responses (see Hennessy and Levine, 1979).

b. *Current Research*

Current research has expanded to include investigations of an integrated, adaptive system of physiological responses to psychological and physical stressors (see Axlerod and Reisine, 1984; Munck *et al.,* 1984; Breazile, 1988; Chrousos and Gold, 1992 for review). Current investigations of physiological stress range from detailed studies of neuroanatomical structures and biochemical changes to the interactions of stress related hormones and their effects on other physiological systems.

i. *Four relevant subsets of current research.* The sources of information for examining responses to stress in this review can be categorized into four general areas of research. First, studies using laboratory or captive animals have investigated responses to stress

under controlled circumstances with stimuli such as electric shock, social isolation, restraint, and administration of exogenous hormones. These types of investigations have lead to information on the neuroanatomical and endocrine pathways of physiological and behavioral responses to stressful stimuli.

Second, investigations assessing stress in domestic animals have become important because of concerns regarding livestock productivity, management practices, and animal welfare. A considerable amount of research has focused on the effects of behavioral, or emotional, stress on livestock (Moberg, 1987a,b, 1996; Friend, 1991; Hemsworth *et al.*, 1993). Relevant studies include controlled investigations of the effects of management related stressors such as handling, transportation, housing, crowding, interspecific social interactions, noise, physical trauma, and psychological factors. Measurements used to assess stress in these studies range from interpretation of behavioral responses to stressors, using cortisol levels correlated with the type, intensity, and duration of the stressor, to assessment of immunocompetence and reproductive function in response to stressors.

Third, clinical research has provided insight into the ways in which humans are affected by stress. In some cases these effects have been shown to be remarkably similar to the ways in which other mammals are affected. Investigation of human subjects with disease states such as Cushing's syndrome (a pathological condition associated with excessive cortisol secretion) has shown the effects of chronic cortisol secretion on the body (Krieger, 1982). Patients suffering from depression exhibit hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis, and studies of these subjects have provided information on the related effects of that activity (Gold *et al.*, 1988a,b). Some athletes

and subjects undergoing forced exercise have been used to investigate the responses of the body to such physical stress (Luger *et al.*, 1987). In addition, longitudinal studies of people who have experienced surgical trauma, illness, war, or even "life events" such as beginning college or a new job, have provided information about human responses to stress (Mason, 1968a; Rabin *et al.*, 1988).

Fourth, studies of free-ranging animals allow us some opportunity to investigate the effects of stress on individuals and, potentially, on populations. In many instances when data have been collected on free-ranging animals, stress had been induced by humans and the stressors included fear, capture, handling, restraint, social separation and displacement from optimal habitat.

In interactions involving humans and populations of free-ranging animals, it is difficult to determine the effects of stress. For example, recent debate surrounds the reasons behind the demise of wild dog, *Lycaon pictus*, populations in the African Serengeti (Burrows *et al.*, 1995; East and Hofer, 1996; East *et al.*, 1997). Burrows (1992) attributed mortality in the wild dogs to research related capture and handling, and suggested that stress induced disease (latent rabies) may have resulted from these practices. To assess the effects of handling on wild dogs, Ginsberg *et al.* (1995) compared rates of mortality and disappearance in handled and unhandled dogs and found no effect of handling on the survival of dogs in any of the five regions studied. Creel *et al.* (1997) measured levels of stress related hormones in fecal samples of radio collared and non-collared wild dogs and concluded that handling and collaring were not persistently stressful for the animals. East (1996) and East *et al.* (1997), have presented

criticisms of the Ginsberg, Creel, and colleagues' studies, and the effects of handling stress in African wild dogs remain undetermined.

These four research areas provide numerous examples of the ways in which mammals respond to various types of stressors, and have led to information regarding the consequences of acute and chronic stress that may be applicable to circumstances in the ETP tuna purse-seine fishery. Studies from each of these areas are used throughout this review to provide indications of the ways in which dolphins interacting with the tuna fishery are likely to respond to stressors.

4. Stress Physiology

Chrousos and Gold (1992) described the mammalian response to stress as a "general adaptational response" that is necessary to an organism's survival and is remarkably consistent in its characteristics among species and even classes of organisms. They described a discrete physiological system that has evolved to coordinate this adaptive response which alters endocrine and neurological function (Chrousos *et al.*, 1988; Chrousos and Gold, 1992) (Figure 1). This adaptive response to stress encompasses both behavioral and physical changes (Stratakis and Chrousos, 1995).

Sapolsky (1998) has compiled an insightful and general overview of physiological responses to stressors. In summary, stress related hormones are released and function to mobilize energy for use by muscle tissue. Heart rate, blood pressure, and respiratory rate increase for efficient transport of oxygen and nutrients to the tissues. Immune function is inhibited, and reproduction, growth, and digestion may be disrupted. Stress-induced analgesia (numbing the body to extreme pain, see Lewis *et al.*, 1980, MacLennan *et al.*,

1982) may occur under some circumstances. Perceptual arousal or alertness is increased, and behaviors such as those related to sex and feeding may be suppressed.

The stress response functions to return the body to homeostasis under conditions of acute, abbreviated stress. Thus, when the duration of a stressor is limited, the physiological effects of stress are beneficial and have no adverse consequences. However, some conditions can lead to dysregulation or pathophysiology resulting from stress (Chrousos and Gold, 1992). Chronic or severe stressors can cause responses that lead to pathologies and the exhaustive state described by Selye (1936).

a. *The Hypothalamic-Pituitary-Adrenal Axis*

The mechanisms by which neuroendocrine changes occur in response to stress are organized primarily in the brain, with hypothalamic secretion of hypophseal releasing hormones and control of the autonomic nervous system. Thus, stress increases sympathetic nervous system activity and the hypothalamic-pituitary-adrenal axis acts to enhance glucocorticoid secretion (see Figure 1).

b. *Corticotropin Releasing Factor and Adrenocorticotropin*

Central to the function of the HPA axis is the release of corticotropin-releasing factor (CRF) from the hypothalamus and adrenocorticotropin (ACTH), from the pituitary, which stimulate synthesis and secretion of glucocorticoids, aldosterone, and adrenal androgens. These neuroendocrine hormones have a significant effect on biochemical, metabolic, and immunological functions, as well as on behavior. Adrenocorticotropin is

also known to have a sensitizing effect on the adrenal cortex, which enhances the response to subsequent stimulation.

Corticotropin-releasing factor is an aminopeptide secreted from the central nervous system, by the hypothalamus (Vale *et al.*, 1981). This aminopeptide hormone has been recognized as playing a major role in regulating the HPA axis because it regulates ACTH secretion and other secretory enhancing agents in the hypothalamus. During stress, levels of CRF secretion increase and cause an concomitant increase in ACTH and cortisol secretion. Also during stressful situations, CRF functions to coordinate a series of adaptive physiological and behavioral responses with the autonomic nervous system (Chrousos, 1992; Chrousos and Gold, 1992). In this manner, CRF coordinates the endocrine response and mediates the metabolic, circulatory, and behavioral adaptations that take place in response to a stressor.

c. *Glucocorticoids*

During stressful events, glucocorticoids act to redirect glucose metabolism from muscle to the brain and other tissues. Glucocorticoids also exert both "permissive" and "regulatory" effects on metabolism, muscle function, cardiovascular function, behavior, and immune function (Brown *et al.*, 1982; Munck and Guyre, 1986; Johnson *et al.*, 1992). During a state of homeostasis, glucocorticoids have a "permissive" action on the activities of norepinephrine and other catecholamines on blood vessels and metabolic pathways, allowing other hormones to function at a normal level, thus maintaining the homeostatic condition (Rivier and Vale, 1983). In a response to stressful events,

glucocorticoid levels are elevated and regulate the reaction of hormones and systems such as the immune system to prevent overreaction which, unchecked, might lead to injury.

d. *Cortisol*

When glucocorticoids are released from the adrenal gland, blood levels of the stress-related hormone, cortisol, increase. Cortisol response (i.e. changes in blood cortisol levels) is often measured to determine the presence and degree of response to a stressor in laboratory investigations. We now know that some percentage of the circulating cortisol is bound to corticosteroid-binding globulin and is physiologically inactive. The amount of bound cortisol varies among species, but can be as high as approximately 95%. Free cortisol is physiologically active, exerting negative feedback to inhibit CRF and ACTH secretion (Siitiri *et al.*, 1982; Johnson *et al.*, 1992). Thus, investigators have begun to measure both total and free cortisol. Moberg (1987a) cautioned that investigators should not rely on cortisol measurements alone as evidence of stress, because some stressors do not elicit an adrenocortical response. Measurements of cortisol levels are often correlated with some other indications of stress such as behavior, heart rate, or serum enzyme levels. Examples of these types of investigations are cited in subsequent sections of this review.

e. *Catecholamines*

Catecholamines, epinephrine and norepinephrine, regulate intermediary metabolism during stress. Secretion of epinephrine and norepinephrine is mediated by preganglionic fibers enervating specialized chromaffin cells within the adrenal medulla

(Greco and Stabenfeldt, 1997). Once secreted, catecholamines cause vasoconstriction, changes in blood pressure and heart rate, as well as metabolic changes. For example, epinephrine increases blood glucose concentrations, mainly through the promotion of hepatic glyconeogenesis, and also stimulates glycogenolysis in skeletal muscle. Epinephrine also causes lipolysis. Catecholamines also stimulate cardiac function, in part by epinephrine's effect increasing the force and contraction of heart rate. In addition, catecholamines affect bronchial, gastrointestinal, and bladder smooth muscle tissue, as well as ciliary muscles of the eyes.

f. *Other Physiological Agents*

In addition to the release of glucocorticoids and catecholamines, there is an increase in the production of other physiological agents or neuroendocrine substances such as angiotensin II, vasopressin, and beta-endorphins (Table 2).

g. *Physiological Stress in Cetaceans*

Cetaceans exhibit the basic mammalian response to stress, and there has been some research investigating the general effects of stress on cetaceans. Many of these studies have focused on assessing blood constituents and stress related hormones. As these reports form the basis of our direct knowledge regarding stress in dolphins, they are outlined below in some detail. There are several physiological responses to stress that have been well documented in cetaceans, including adrenocortical responses and effects on thyroid hormone balance. Each of these is discussed in turn below.

i. *adrenocortical response.* There are two notable aspects of the adrenocortical response to stress in cetaceans. First, although elevated cortisol levels are evident in cetaceans subjected to stressors such as capture, handling, and restraint, these elevations appear to be modest in comparison to those known for other mammals experiencing similar stressors (Thomson and Geraci, 1986; St. Aubin and Geraci, 1990). St. Aubin and Geraci (1990) noted that, despite this apparently modest response, the systemic effects of cortisol are still evident in decreased circulating levels of eosinophils and reduced plasma iron, as well as in increased levels of glucose (as described below). Second, aldosterone, not typically characterizing the adrenal response to stress in terrestrial mammals, is greatly increased in cetaceans subjected to adrenocortical stimulation (Thomson and Geraci, 1986; St. Aubin and Geraci, 1990). An increase in aldosterone has also been observed in response to adrenal stimulation in pinnipeds (St. Aubin and Geraci, 1986). Aldosterone functions to enhance water and sodium reabsorbtion, so the response observed for cetacean and pinniped species studied thus far is thought to reflect the necessity of regulating these processes during stress (St. Aubin and Geraci, 1986; St. Aubin *et al.* 1996).

ii. *Thyroid hormone balance.* Thyroid hormone balance in cetaceans appears to be sensitive to stress. Ridgway and Patton (1971) found that several captive Pacific white-sided dolphins, *Lagenorhynchus obliquidens*, had low plasma levels of thyroid hormones and suggested that chronic stress in cetaceans could depress thyroid function. St. Aubin and Geraci (1988) found that thyroid hormone levels were suppressed during capture stress in free-ranging beluga whales, *Delphinapterus leucas.* The authors measured

plasma concentrations of triiodothyronine (T3) and thyroxine (T4), in 24 captured juveniles. For animals detained at the capture site, concentrations of T3 decreased significantly during the first 24 hours and after two to four days there was also a reduction in T4. Concentrations of T3 and T4 remained suppressed during the ten weeks of captivity. Injection of ACTH resulted in a further decrease in T3 after 6-12 h. Handling stress alone produced similar changes in individuals injected with saline as a control. St. Aubin and Geraci (1992) also found decreases in thyroid hormone levels in beluga whales subjected to capture, and Orlov *et al.* (1988) reported decreases in T3 as the result of handling captive bottlenose dolphins. Although chronic thyroid hormone imbalance can have deleterious effects on growth and metabolism, the changes seen in these studies of captive cetaceans have been interpreted as adaptive responses to glucocorticoids, acting to realign thyroid hormone balance (St. Aubin and Geraci, 1988, 1992; and see St. Aubin *et al.,* 1996).

iii. *Capture stress and cetaceans.* Cetaceans can be affected by the stress of capture. Several studies have contributed to our understanding of their physiological responses to capture stress and adrenocortical stimulation with exogenous ACTH (Thomson and Geraci, 1986; St Aubin and Geraci, 1989; St. Aubin *et al.*, 1996). For example, Thomson and Geraci (1986) measured circulating cortisol, aldosterone, and eosinophils in the blood of captive bottlenose dolphins subjected to net capture and restraint. Two capture conditions were employed: 1. "calm-capture" in which a dolphin was netted within 10 minutes with as little herding as possible, and 2. "chase-capture" in which a dolphin was

repeatedly chased, captured and released. Blood samples were collected 10 minutes after capture, 15 minutes after removal from water, and at 30 minutes, 1, 3, and 6 hours thereafter. Following calm-capture (during restraint) serum cortisol increased from resting levels of about 30nmol/L to 110nmol/L within the first hour and remained within this range for the remaining 6 hours, aldosterone increased from approximately 280pmol/L to 1880pmol/L within three hours, and circulating eosinophils were less than 40% of their initial values within seven hours. Blood cortisol levels were reported to be 100% higher for dolphins immediately after chase-capture, when compared to samples collected immediately after calm-capture. Thereafter, the basic response was not enhanced (Thomson and Geraci, 1986). The observed decrease in eosinophils is considered to be important because these white blood cells function in response to allergies and infection. Thomson and Geraci (1986) concluded that eosinophil counts are consistent and practical indicators of stress in dolphins.

It is interesting that the cortisol response was not enhanced during prolonged (3 hours) capture, or upon administration of exogenous ACTH (Thomson and Geraci, 1986). This was surprising because the ACTH challenge induces maximum levels of glucocorticoid secretion by the adrenal cortex in most mammals. In cetaceans, cortisol secretion does not appear to increase in response to exogenous ACTH to levels higher than those observed during capture stress (Thomson and Geraci, 1986; St. Aubin and Geraci, 1990). In their study, Thomson and Geraci (1986) suggested that adrenal cortisol secretion might have been influenced by preexisting cortisol levels that had been induced by the initial stress response of bottlenose dolphins to capture and handling. As noted

previously, in contrast to terrestrial mammals, aldosterone levels do rise in response to ACTH challenge in cetaceans (Thomson and Geraci, 1986; St. Aubin and Geraci, 1990).

Capture was also found to increase levels of aldosterone, cortisol, glucose, iron, potassium, and the enzymes creatine kinase (CK), aspartate aminotransferase (AST), alanine aminotransferase (AP), and gamma-glutamyltranspeptidase (GGT), in 24 live captured beluga whales (St. Aubin and Geraci, 1989). Changes in leucocytes were also reported for these animals, including acute lymphopenia, eosinopenia, and mild neutrophilia. No details of capture methods were given, but approach and capture was reported to take less than 30 minutes. Capture lasted for up to ten weeks, and most indices were reported to normalize within the first week of captivity.

In addition, muscle activity during pursuit can affect muscle enzyme (CK and AST) levels (St. Aubin and Geraci, 1989). This may reflect muscle damage. Elevated levels of CK and potassium were suspected to be related to possible muscle damage in an adult female bottlenose dolphin subjected to enforced movement and chemical intoxication. Over a 72 hour period, the dolphin was subjected to continuous movement and "nudging," and was held in a shallow pool from which it was periodically removed for blood sampling. This animal also exhibited elevated glucose levels, which were considered to result from glycogen mobilization related to stress (Geraci and Medway, 1973).

Several blood parameters associated with muscle damage in mammals were reported for net caught dolphins in South Africa (Young *et al.*, 1997). Blood serum levels were analyzed for captive bottlenose dolphins, *Tursiops* spp. and incidentally caught (free-ranging) common, Indo-Pacific hump-backed, *Sousa chinensis*, and

bottlenose dolphins in South Africa. Values were pooled across species for free-ranging individuals sampled, and not all blood parameters were analyzed for all samples. Mean values of most parameters measured were elevated in the free-ranging animals that had been captured. In particular, lactate dehydrogenase (LDH), CK, and creatinine were elevated. These values are likely to indicate muscle damage, but are difficult to interpret because data for individuals were not presented. Young *et al.* (1997) considered their results to indicate that the entangled dolphins experienced severe stress, but methods and information regarding post-mortem blood sampling and conditions were not described in sufficient detail to test the validity of their conclusions.

Recently, St. Aubin *et al.* (1996) measured circulating hormone levels in 36 free-ranging bottlenose dolphins, sampled from a coastal population, and 36 "semidomesticated" bottlenose dolphins. Small groups of free-ranging dolphins were encircled with a seine net using a high-speed motor boat. These dolphins were manually restrained and placed in a boat for examination. Semidomesticated dolphins had been conditioned to voluntarily present their flukes for blood sampling. Cortisol, aldosterone and thyroid hormones (thyroxine (free (fT4) and total (tT4)), triiodothyronine (free (fT3), total (tT3), and total reverse (rT3)) were measured. The samples were matched by age, sex, and time of year collected. Patterns of thyroid hormones levels varied, and appeared to be influenced by age and sex. The most consistent difference between the free-ranging and semidomesticated animals was in adrenal hormone levels (St. Aubin *et al.*, 1996). Levels of cortisol and aldosterone were low in semidomesticated dolphins, and the collection technique was acknowledged to produce samples representative of resting values for these constituents (St. Aubin *et al.*, 1996). Cortisol and aldosterone were

higher in free-ranging animals sampled after a variable interval of up to four hours after encirclement by capture net. The authors considered this pattern to represent a mild stress response.

In summary, the few available data on physiological effects of capture in cetaceans indicate that the process does elicit a stress response (activation of the HPA axis; Thomson and Geraci, 1986; St Aubin *et al.,* 1996). Capture stress can also apparently cause muscle damage (Geraci and Medway, 1973; St. Aubin and Geraci, 1989) and changes in thyroid hormone balance in cetaceans (St. Aubin and Geraci, 1992; St Aubin *et al.*, 1996). Several effects related to immune function (changes in blood leukocytes and decreases in blood iron levels) have also been observed in captured cetaceans. Information regarding the effects of repeated capture is not available. It is possible that repeated capture could exacerbate stress and elicit an anticipatory response as has been suggested for some species such as captive impala, *Aepyceros melampus.* Hattinagh *et al.* (1988) compared the physiological response of captive and free-ranging impala to repeated capture, and found that blood cortisol levels were significantly higher in captive impala (93 $\pm$21 nmol/l; free-ranging, 11 $\pm$3 nmol/l, $p<.05$). The authors suggested that higher cortisol levels in captive impala resulted from a conditioned anticipatory response occurring prior to capture (Hattinagh *et al.,* 1988). There may be some evidence for this from the scientific literature on cetaceans. Newly captured beluga whales may have had an anticipatory response to sampling procedures, in so much as each time the water level in their holding pool was lowered it appeared to elicit pulsatile release of cortisol (30 $\pm$22nmol/L higher than samples drawn 5-6 hours previously; St.

Aubin and Geraci, 1992). Alternatively, repeated capture could also result in acclimation or habituation in cetaceans.

There is some indication that dolphin behavior has changed since the early years of the fishery, and that this may be the result of learning or habituation[1]. Limited information suggests that dolphins in areas with high historical fishing effort are more experienced at avoiding chase and encirclement than more naive dolphins in areas where fishing had expanded more recently. Fishery observers have noted that dolphins apparently anticipate backdown because they move to the backdown channel and "line-up" for release.

Still, the semidomesticated dolphins in the St. Aubin *et al.* (1996) study had been in captivity for up to 30 years and were "accustomed" to presenting their flukes for husbandry techniques. In addition, Thomson and Geraci (1986) found that even dolphins that had been routinely handled for years in captivity had a stress response after the calmest capture conditions possible. Habituation such as that of the semidomesticated dolphins studied by St. Aubin *et al.* (1996) does not seem likely in free-ranging animals such as dolphins in the ETP. In fact, given the tendency of herds in the ETP to avoid approaching vessels, the occurrence of an anticipatory stress response in these animals seems more probable.

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[1] A study regarding "evasive behavior" of spotted dolphins was completed by G. Heckel, Universidad Autonoma Baja California, Baja California, Mexico, and has been summarized in the NMFS Tuna Newsletter, November 1997.

II. THE POTENTIAL FOR FISHERY-INDUCED STRESS ON DOLPHINS IN THE ETP

The potential for fishery-induced stress to have adverse effects on dolphins in the ETP has been a concern since at least the late 1970's when Norris *et al.* (1978) suggested that every aspect of purse-seine capture causes stress to the dolphins involved. In assessing the potential effects of search, chase and capture on dolphins in the ETP, it is important to acknowledge that physiological responses to stress may be adaptive and temporary, acting to restore homeostasis. Such physiological changes may be of little consequence to individual dolphins. It does not appear possible to predict the specific level or duration of changes in physiological parameters that could become adverse to the health of individual dolphins.

Some of the plausible consequences of potential fishery-induced stress on dolphins are discussed below in connection with the various aspects of fisheries operations that are hypothesized here to be potentially stressful for the dolphins involved. Some potential consequences of the search are described first, followed by consideration of potential short and long-term effects of chase and capture. Long-term pathological responses to stress are discussed in the context of potential fisheries-induced dysregulation of physiological systems. Information was derived from several direct studies of spotted and spinner dolphins involved in the fishery, and from information about responses to stress in other mammals.

A. POTENTIAL CONSEQUENCES OF SEARCH

At some point during search the dolphins become aware of the helicopter and the purse-seine vessel, and their reaction is to move away (see Norris *et al.*, 1978). Dolphins in the ETP apparently avoid approaching ships (Norris *et al.*, 1978; Au and Perryman, 1982; Hewitt, 1985). This is potentially significant in terms of indicating the herds' perception of the fishery (i.e. the dolphins may associate vessels with the subsequent chase and capture). Norris *et al.* (1978) reported that dolphins reacted to the helicopter even at elevations of 700-1000 feet above them.

Helicopter flights are known to influence movements in terrestrial mammals. For example, it has been concluded that helicopter flights could have an adverse impact on populations of bighorn sheep, *Ovis canadensis*, by altering movements, habitat use, and foraging efficiency and thus reducing survivorship and reproduction (Stockwell *et al.*, 1991; Bleich *et al.*, 1994). Specifically, Stockwell *et al.* (1991) found that disturbance from helicopter overflight diminished foraging efficiency in bighorn sheep. In addition, Bleich *et al.* (1990) found that spatial distribution of bighorn sheep was substantially altered by disturbance during helicopter surveys. Of relevance here is that the sheep did not become habituated or sensitized to frequent helicopter flights (Bleich *et al.*, 1994).

B. POTENTIAL CONSEQUENCES OF CHASE AND CAPTURE:

IMMEDIATE EFFECTS

1. Psychosocial Effects of Chase and Capture

The following sections address potential psychological or social stimuli associated with the stress of purse-seine capture and the likely physiological responses to

these stimuli. The perception of control over and predictability of stressors may influence the occurrence of stress-related disorders (Friend, 1991), and this may act as one underlying psychological stressor influencing the physiological stress response to chase and capture. For example, a dolphin's perceptions of its ability to avoid capture or its inability to escape may play a role in the degree of physiological response to the event. Psychosocial stressors are remarkably potent stimuli for activation of the HPA axis (see Mason, 1968a) and are likely to be of importance in characterizing the stress response in ETP dolphins.

a. *Social Separation*

Disruptions of social groups, including separation of closely associated conspecifics such as cow-calf pairs could occur as a result of chase and capture. Based on information from other mammals, these types of disruptions are likely to cause physiological stress (activation of the HPA axis) in individual dolphins.

It is well known that social relationships influence physiological function (e.g. Hinkle, 1974). For mammals, social relationships and the presence of familiar social partners are an important factor in determining the magnitude of the endocrine response to stress (Levine, 1993). For example, the presence of a familiar social partner, particularly in the case of the mother-young relationship, can modulate cortisol response to a stressor. In domestic goats (Lyons *et al.*, 1988b) and squirrel monkeys, *Saimiri sciureus* (Mendoza *et al.*, 1978), presence of the mother moderates or can even eliminate a young animal's cortisol response to stressors. The presence of a surrogate mother has

also been demonstrated to moderate the cortisol response in squirrel and rhesus monkeys (Hill *et al.*, 1973; Mendoza *et al.*, 1978).

In contrast, separation from familiar conspecifics can elicit a physiological stress response. This appears to be particularly true in the case of the mother-young relationship (Levine, 1993). In laboratory studies, disruptions of mother-young relationships have activated the HPA axis (measured as elevated plasma cortisol levels) in both mother and infant (Levine and Weiner, 1989; Weiner *et al.*, 1990). In rodents, two hours of maternal deprivation leads to significant increases in basal cortisol levels, and longer periods elicit progressively greater increases (Levine *et al.*, 1991). In addition, the response may not decrease over time, even after repeated events. For example, in squirrel monkey mother and infants subjected to multiple disruptions, no physiological or behavioral indications of habituation were evident over time (Hennessy, 1986).

Separation of mother and infant rats has been shown to cause elevated cortisol levels (Khun *et al.*, 1990; Levine *et al.*, 1991; Rosenfeld *et al.*, 1991). In addition, the response appears to be related to an increased sensitivity of the adrenal gland because deprived infant rats have a greater cortisol response to ACTH than do non-deprived infants (Levine *et al.*, 1991; Rosenfeld *et al.*, 1991). Suchecki *et al.* (1993) investigated maternal deprivation of infant rats, and found that ACTH levels were elevated in infants deprived of their mothers. The authors hypothesized that removal of the mother elicits an increase in synthesis and traffic of neuropeptides (CRF and vassopressin), thus enhancing ACTH secretion. They suggested that factors of maternal origin have some influence on the infant ACTH response to stress (Suchecki *et al.*, 1993).

Beyond the mother-young relationship, social groups are also important in moderating responses to stressful situations for all age classes (Levine, 1993). To test this, Coe *et al.* (1982) exposed squirrel monkeys, both individually and in their social groups, to the presence of a snake. In both cases, the monkeys' behavioral responses were similar (they became visibly agitated) but when accompanied by group-mates there was no cortisol response.

b. *Isolation and Restraint*

Isolation and restraint stress is relevant here to the extent that a dolphin separated from its mother, from closely associated conspecifics, or from an entire social group, while confined in a purse-seine net, may perceive its condition to be one of isolation and restraint. Although there may be differences between actual physical restraint and confinement in a relatively broad space, confinement of any form may be especially stressful for these pelagic dolphins because they are unaccustomed to encountering physical barriers of any kind in their open ocean environment.

Isolation and restraint can cause stress in mammals. Isolation elicits increased cortisol levels in lambs (Moberg *et al.*, 1980; Niezgoda *et al.*, 1987), and combined restraint and isolation caused sustained high levels of cortisol in lambs (Minton and Blecha, 1990). Niezgoda *et al.* (1987) found that three treatments of repeated isolation (5 hours) of lambs from the flock spaced at 3 day intervals, caused increased levels of cortisol with each successive treatment. Minton and Blecha (1990) sampled blood at 15 minute intervals, from lambs subjected to combined restraint and isolation stress and observed an immediate fourfold increase in cortisol levels compared to control lambs.

Cortisol levels gradually decreased after treatment, but remained elevated in comparison to the control group even at the last sampling, 18 hours after release from isolation and restraint. Cortisol levels in lambs subjected to restraint and isolation have remained elevated even after three consecutive days of application (Minton *et al.*, 1992). Moreover, it has been shown that the repeated application of restraint and isolation stress compromised immune response to mitogens (Coppinger *et al.*, 1991; Minton *et al.*, 1992) and alterations of leukocyte differentiation antigens in lambs (Minton *et al.*, 1992). The topic of impaired immune function in relation to stress is discussed more fully below (see section II.C.2, Stress-Induced Changes in Immune Function).

c. *Social Aggression*

During purse-seine capture, dolphins are crowded together, possibly without sufficient space to avoid interaction with individuals they might otherwise be naturally separated from in the social organization of the herd. In their study of dolphin behavior in tuna purse-seine nets, Pryor and Kang Shallenberger (1991) observed that social groups tended to stay tightly together in so far as it was possible while confined in the nets.

Aggressive behavior between domestic animals placed in crowded conditions, or free-ranging conspecifics placed in captive situations where established dominance hierarchies are disrupted have been widely documented. Subordinates may find themselves in close and inescapable proximity to higher ranking individuals, thus subjected to the stress that is associated with low rank in some social hierarchies (Sapolsky, 1985). In addition, Sapolsky (1987a) suggested that in times of social

instability, social aggression is associated with high rank and, at least in the case of males, with initial high levels of testosterone and glucocorticoids.

d. *Novelty*

Novelty can be defined as something new or unusual. There are few data regarding capture frequency of dolphins in the ETP tuna purse-seine fishery. Chase and capture are likely to be novel to at least some (neonatal/juvenile) portion of the population at any given time. Moreover, in cases where herds have not encountered vessels for some time, introduction of this stimulus may in itself provide a change of environment sufficient to be considered novel.

Novelty is relevant to the question of stress effects on dolphins in the ETP because novel situations can activate the HPA axis resulting in elevated levels of circulating glucocorticoids. For example, in mice and rats, the novelty of being placed in a new chamber can elicit corticosteroid levels as great as those measured in individuals that have received electric shock in the chamber (Friedman and Ader, 1967; Bassett et al, 1973; Hennessy *et al.*, 1977). Also, squirrel and titi, *Callicebus moloch*, monkeys exposed to a seemingly minor novelty (transfer into an otherwise identical cage) had significant and sustained (lasting more than two days) elevations in plasma cortisol (Hennsessy *et al.*, 1995).

However, differences in temperament as well as experience appears to influence any particular individual's response to novel situations. In addition, some elements of stability (e.g. in environment or social relationships) are important moderators of an individual's response to challenge. For example, both genotype and early postnatal

environment are important in the development of stable individual differences in temperament and response to challenge in domestic goats (Lyons *et al.*, 1988a). Hennessy *et al.* (1995) demonstrated different physiological responses to novelty between squirrel and titi monkeys, and suggested that the differences might be related to certain socioecological differences between the two taxa.

This information implies that there could be taxonomic differences between spotted and spinner dolphins in the physiological responses to psychosocial stressors. In addition, it can be expected that there will be individual differences among dolphins in the ETP which are likely to be manifested in individual variation in the nature and degree of physiological response to stress.

The available evidence also implies strongly that disruption of the mother-young bond in ETP dolphins will activate the HPA axis for both mother and infant. Further, disruption of social groups is likely to cause this physiological stress response among some proportion of individuals in all age classes. This stress response, activation of the HPA axis, in itself may serve as an adaptive mechanism for the individual to achieve homeostasis. In repeated acute or chronic cases, however, activation of the HPA axis could be detrimental as discussed below (see Long-Term Effects).

2. Immediate Physiological Effects Related to Chase and Capture

Forced exercise during chase is a significant part of the purse-seine capture procedure for dolphins in the ETP. Speed, distance and duration of the chase are all factors that are likely to affect the dolphins' responses to stress. The extent to which chase in the ETP fishery affects the respiratory, muscle, and thermoregulatory capacities

of dolphins is not known. It has been shown, however, that dolphins are exceptional swimmers. Williams *et al.* (1992) illustrated the low cost of transport for dolphins relative to other swimming animals, noting that hydrodynamic shape and economy of movement may contribute to the dolphin's efficiency as a swimmer. This may imply that these pelagic dolphins are predisposed to endure long bouts of aerobic exercise.

Based on previous investigation of delphinid muscle tissue, however, it is not unreasonable to expect that muscle activity could be affected by chase. Studies of bottlenose dolphin muscle fiber type, size, and arrangement indicate a 50:50% (fast twitch:slow twitch) composition of swimming musculature signifying a capacity for brief, rapid contractions, and continuous, slower contractions (Goforth, 1984). Muscle damage resulted from experimental capture of captive bottlenose dolphins (Thomson and Geraci, 1989), and therefore it seems reasonable that muscle damage may also occur in dolphins as a result of chase and capture in the ETP fishery.

Muscle damage can result from strenuous exercise in mammals (Boyd, 1982; Janssen *et al.*, 1989; Armstrong, 1990). During muscle disruption, muscle enzymes leak into the blood via alteration of cellular membranes and tissue damage (Janssen *et al.*, 1989). Increased serum plasma levels of the enzyme CK, which is known to catalyze the exchange of energy during muscle activity (Conley, 1994), is a reliable indicator of muscle tissue damage (Noakes, 1987; Hortobagyi and Denahan, 1989; Volfinger *et al.*, 1994).

In a recent study on the effects of pursuit on hunted red deer, *Cervus elaphus*, blood and muscle samples were analyzed to assess possible muscle damage resulting from the hunt (Bateson, 1997; Bateson and Bradshaw, 1997). Blood and muscle samples

were collected from 64 hunted and 50 non-hunted (cleanly shot) deer. A number of blood plasma constituents were measured, including the muscle enzymes CK, LDH, and AST. These muscle enzyme levels were higher in blood samples from hunted deer, indicating muscle disruption. Bateson and Bradshaw (1997) related the muscle damage to a condition known as "capture myopathy."

a. *Capture Myopathy*

Capture myopathy results from muscle exertion associated with capture and restraint of wildlife. Myopathies are considered to be diseases of the muscle fiber, and there are several types including: congenital, nutritional, toxic, and capture myopathies (Hadlow, 1973; Heldstab and Rüedi, 1980).

Capture myopathy has been described in numerous mammal and bird species (Chalmers and Barrett, 1977; Basson and Hofmeyr, 1978; Carpenter *et al.*, 1991; Dabbert and Powell, 1993; Bailey *et al.*, 1996; Williams and Thorne, 1996). The condition results from a wide variety of capture situations, including chase by vehicles and aircraft[2], capture in traps or nets, drug induced immobilization, and restraint (Harthoon and Young, 1974; Colgrove, 1978; Gericke, *et al.*, 1978; Wallace *et al.*, 1987; Kock *et al.*, 1987b; Hattingh, 1988) (Tables 3a,b).

Capture myopathy is characterized by a variable and lengthy list of clinical signs including ataxia, paralysis, myoglobinuria, and acute muscle degeneration (Hulland,

[2] Chase is not always a component of capture in documented cases of capture myopathy, and the condition can occur as the result of capture alone. Duration of chase that has been reported in association with capture myopathy is quite variable, and little information is available on the speed or intensity of chases associated with the condition. Often, helicopters or other aircraft have been used in documented cases of capture myopathy that have occurred in association with chase and capture (see Table 3b).

1985; Harthoon and Young, 1974; Bartsch *et al.*, 1977; Chalmers and Barrett, 1977; Basson and Hofmeyr, 1978). General clinical indications of capture myopathy include elevated blood serum enzymes (AST, CK, LDH), and blood urea nitrogen (BUN) levels. Initial clinical symptoms of the condition include increased respiratory and cardiac rates and elevated body temperature. The latter is important clinically because the development of capture myopathy appears to be associated with hyperthermia (Williams and Thorne, 1996). Other early signs include lack of responsiveness to the environment, weakness, and muscle stiffness. Paralysis, most often observed to occur in the locomotory muscles of terrestrial mammals, may develop. Lesions are also often observed from histological examination of animals diagnosed with capture myopathy (Table 3a).

Spraker (1993) provided a concise overview of capture myopathy in animals and considered four clinical syndromes to be associated with the condition. These include capture shock, ataxic myoglobinuric, ruptured muscle, and delayed-peracute syndromes. Spraker (1993) suggested that capture myopathy can be induced by a combination of many stressors (e.g. terror, chase, capture, restraint), and that it is associated with exhaustion of the normal physiological reserves that provide energy for escape. Time to exhaustion is dependent upon the individual species, the stressor itself, and environmental conditions (Spraker, 1993).

It is entirely plausible that the delayed-peracute syndrome described by Spraker (1993) could occur in dolphins involved with the ETP fishery. Clinical signs characterizing this syndrome have been described in numerous cases for terrestrial mammals and do not develop for hours or even weeks after capture (see Chalmers and

Barrett, 1977; Basson and Hofmeyr, 1978; Williams and Thorne, 1996). Spraker (1993) noted that in the case of delayed-peracute syndrome, animals appear normal when undisturbed, but subsequent disturbance, repeated capture or additional stress is likely to cause immediate death.

Cowan and Walker (1979) examined 49 spotted and 16 spinner dolphins killed during routine fishing operations in the ETP fishery. They examined several primary locomotor muscles (Longissimus dorsi and Hypaxial muscles) for both gross pathological or histopathological changes. No evidence of myopathy was detected in the skeletal muscles of the 56 animals examined (Cowan and Walker, 1979). However, gross lesions may be absent in the delayed-peracute capture myopathy syndrome, and histological lesions associated with the syndrome may be characterized as mild or moderate (Spraker, 1993).

Stuntz and Shay (1979) reported the results of a workshop held to investigate the potential occurrence of capture myopathy in dolphins involved in the ETP fishery. Although no data were provided, Stuntz and Shay (1979) reported that blood samples had been collected from 34 dolphins captured in the fishery and that at least a portion of those animals had elevated blood CK levels. The elevated values were considered to indicate "muscular exertion or muscle damage," but participants agreed that further information on laboratory techniques and controls as well as on delphinid physiology were necessary to interpret the findings (Stuntz and Shay, 1979). The general consensus of the workshop was that it was likely that some unobserved mortality was occurring after dolphins were released from the nets.

In summary, capture myopathy affects the physiological status of birds and mammals, and influences post-capture survival (Harthoon and Young, 1974; Chalmers and Barrett, 1977; Dabbert and Powell, 1993). It is likely that, when released, animals suffering from capture myopathy are predisposed to predation and accident (Williams and Thorne, 1996). For example, it has been noted that death subsequent to capture of waterfowl can be "delayed or indirect" and could bias population studies that assume natural mortality rates for released animals (Bollinger *et al.*, 1989; Dabbert and Powell, 1993).

b. *Hyperthermia*

It is possible that dolphins experience some degree of hyperthermia resulting from being chased and herded by speedboats in the ETP. Hyperthermia caused by chase and capture has deleterious effects in mammals (Spraker, 1980), and appears to be associated with the development of capture myopathy (Chalmers and Barrett, 1982; Antognini *et al.*, 1996; Williams and Thorne, 1996).

Generally in mammals, if heat balance is not achieved several complications may arise. Continuous elevations in body temperature can have adverse effects on muscle metabolism and the central nervous system (MacDougall *et al.*, 1974; Brinnel *et al.*, 1987; Koroshetz and Bonventure, 1994). Muscle temperature rises most rapidly during strenuous exercise, with core temperatures increasing subsequent to rise in blood temperature (Jones *et al.*, 1989). While temperatures causing fatigue are not clear, the safe maximum temperature for muscle tissue in most mammals appears to be approximately 45° C (Hodgson *et al.*, 1994). Maximum temperature reached in dolphins during chase in the ETP fishery are not known.

It is known, however, that the injurious effects of hyperthermia resulting from strenuous exercise can be severe. For example, hyperthermia caused by prolonged exercise in horses can result in exertional rhabdomyolysis (myopathy), synchronous diaphragmatic flutter, ataxia, and collapse (Hodgson *et al.*, 1994). Severe cases of hyperthermia can result in depression, coma and death (Spraker, 1980; Hodgson *et al.*, 1994).

Hyperthermia can also impair reproduction in mammals. Bell (1987) reviewed the effects of maternal hyperthermia caused by factors such as environmental heat exposure, fever, and strenuous exercise at different stages of pregnancy. Effects in early pregnancy include embryo mortality, teratogenesis (development of fetal abnormalities), and possibly impaired learning abilities (Jonson *et al.*, 1976; Edwards, 1986; Bell, 1987). During middle to late pregnancy, maternal hyperthermia can cause fetal growth retardation. Bell (1987) has suggested that placental insufficiency (changes in placental metabolism and growth) is the ultimate cause of fetal growth retardation associated with chronic maternal hyperthermia. Based on these studies in other mammals, it is reasonable to assume that hyperthermia could impose deleterious effects on female reproduction in cetaceans. In fact, decreased blood flow to the uterus (resulting from increased demand of locomotory muscles or shunting blood to eliminate excess heat) and fetal exposure to increased temperatures in cetaceans are likely to expose the fetus to the deleterious developmental effects described above (Rommel *et al.*, 1993).

Hyperthermia can also affect reproduction in the male. Heat reduces semen quality in domestic animals (Marschang, 1973; Larsson and Einarsson, 1984; Stone 1981). Cetacean testes are located within the abdominal cavity, and positioned close to

axial and abdominal muscles where their temperatures may be affected by core body temperatures (Boice *et al.*, 1964; Rommel *et al.*, 1992). An arterio-venous countercurrent heat exchanger associated with the testes may regulate temperature via blood flow and diminish the detrimental effect of high temperatures on sperm viability and spermatogenesis (Rommel *et al.*, 1992). The potential effects of hyperthermia on male reproduction in cetaceans remain undetermined.

Information from other mammals suggests that hyperthermia could result from chase and capture of dolphins in the ETP. There have been only a few estimates of the thermoregulatory condition of spotted and spinner dolphins (Hampton and Whittow, 1976; Worthy and Edwards, 1990). For spotted dolphins collected in the ETP tuna fishery, surface area/volume ratio was not significantly different than for terrestrial mammals (Worthy and Edwards, 1990). Heat balance during strenuous exercise could be facilitated through the heat exchangers in the flukes and fins (Scholander and Scheville, 1955; Brodie, 1975). While there is some information regarding temperature regulation in marine mammals (see Whittow, 1987; Hokkanen, 1990, for review), little is yet known about the effects of forced strenuous exercise on thermoregulation in these animals. It is possible, but as yet undetermined whether chase and capture could negatively affect thermoregulation in dolphins involved in the tuna fishery.

3. Capture Stress and Noise

It is also possible that fishery-related noise causes stress in dolphins in the ETP. Dolphins captured in the tuna purse-seine fishery are exposed to noise from helicopters, as well as from speedboat and purse-seine vessel engines. However, the effect of noise

on marine mammals is not extensively known (but see Jefferson and Curry, 1994; Richardson *et al.*, 1995; Ketten, 1998). Thomas *et al.* (1990) evaluated the response of captive beluga whales to drill-rig noise. The authors did not observe elevated levels of blood catecholamines in response to the noise and determined it did not cause stress to the animals (Thomas *et al.*, 1990). There is insufficient information on the type, intensity, and duration of noise that dolphins are exposed to in the ETP fishery, to surmise the potential effects of noise on the animals.

C. POTENTIAL CONSEQUENCES OF CHASE AND CAPTURE: LONG-TERM EFFECTS

1. Stress-Induced Pathologies

Dysregulation of the HPA axis resulting from severe acute or chronic stress can result in structural, as well as, systemic pathologies. For example, glucocorticoids induce a catabolic effect on peripheral organs (skin, muscle, and adipose tissue), promoting protein and lipid degradation (Junqueira and Carneiro, 1971). Chronic stress also causes hypertrophy and hyperplasia of the adrenal cortex and medulla (Anderson and Capen, 1978; Harvey *et al.*, 1984). Stress affects thyroid function and can cause morphological changes in that gland. In addition, glucocorticoids promote hepatic gluconeogenesis and the increased metabolic activities of the liver can lead to hyperplasia in that organ under conditions of chronic stress.

Stress can also cause cardiomyopathy, a condition characterized by focal myocardial necrosis induced by stressful stimuli. Cardiomyopathy has been documented in rats (Raab *et al.*, 1968), squirrel monkeys (Corley et al, 1973, 1975), pigs (Johansson

et al., 1974), and humans (Cebelin and Hirsch, 1980). The lesion may be caused by catecholamines released in response to acute stress (Reichenbach and Benditt, 1970).

a. *Stress-Related Pathologies in Dolphins*

Two studies have investigated the occurrence of stress-related pathologies in specimens of spinner and spotted dolphins recovered from the ETP fishery. In the first study, Cowan and Walker (1979) examined spotted and spinner dolphins killed in the ETP fishery and defined three states of disease: 1. naturally occurring disease, 2. tissue changes resulting from acute responses to terminal events, and 3. subacute pathological conditions that could hypothetically lead to death. Parasitism was found to be the most prevalent cause of naturally occurring disease. Acute tissue changes were attributed to drowning in fishing and research gear, and were described for the lungs, heart, adrenal glands, and spleen.

Cowan and Walker (1979) concluded that there was no substantial evidence of "delayed mortality" related to the fishery (but see section II.B.2.a, Capture Myopathy). However, they noted that several of the dolphins apparently died of massive cardiac reaction to stress and were documented to have cardiac lesions consistent with those produced in laboratory animals injected with catecholamine and humans thought to have died of stress cardiomyopathy (Cowan and Walker, 1979; and see Cebelin and Hirsch, 1980).

In the second study investigating the potential occurrence of stress-related pathologies in dolphins killed in the ETP fishery, the adrenal glands of 90 male spinner and 172 male spotted dolphins were examined with the hypothesis that "continuous acute

stress" caused the deaths of dolphins caught in purse-seine nets (Myrick and Perkins (1995). Thirty-nine histological sections were examined for lipid content and erythrocyte leakage, but adrenal color was used as the primary method to assess the presence of lipids and congestion in the glands. The authors concluded that the darkened cortices observed in approximately 95% of the samples indicated continuous acute stress and/or vasogenic shock. However, color is a volatile character and could also present artifacts (autolysis or pooling of blood postmortem, for example) to the analysis, thus the results must be regarded with caution.

Adrenocortical cysts, which may be another stress-related pathology, have been observed to occur in several cetacean species including in mass stranded Atlantic white-sided dolphins, *Lagenorhynchus acutus* (Geraci *et al.*, 1978), stranded harbor porpoises, *Phocoena phocoena* (Kuiken *et al.*, 1993), a captive common dolphin (Cartee *et al.*, 1995), and stranded beluga whales (Lair *et al.*, 1997). The mass stranded Atlantic white-sided dolphins could be considered to represent a generally "healthy" population, and Geraci *et al.* (1978) determined that the adrenocortical cysts observed in those animals, were probably stress-related. Adrenocortical hyperplasia was observed in 24 out of 35 stranded belugas, and cortical cysts containing a cortisol concentrated fluid were found in 19 of the whales (Lair *et al.*, 1997). The belugas sampled were exposed to xenobiotics and generally suffering from chronic disease. Lair *et al.* (1997) suspected that the adrenocortical cysts might have been stress-related. Harbor porpoises that were also chronically sick and exposed to chlorinated hydrocarbons were observed to have adrenocortical hyperplasia that was interpreted to result from chronic stress (Kuiken *et al.*, 1993).

2. Stress-Induced Changes in Immune Function

Stress also can have significant effects on immune function. Secretion of adrenal glucocorticoids induced by stress limits immune and inflammatory reactions through glucocorticoid induced changes in the traffic and function of immune system cells (Munck *et al.*, 1984; Breazile, 1987, 1988; Dantzer and Kelley, 1989; Munck and Guyre, 1991; Chrousos and Gold, 1992; Chrousos, 1995). Glucocorticoid hormones reduce blood leukocytes (especially helper T lymphocytes, monocytes, and eosinophils) and decrease the clonal generation of lymphocytes (natural killer cells). These compromises in immune function lead to impaired response against tumor and virus-infected cells. Natural killer cell activity, for example, provides non-specific defense against viral infections and tumors (Breazile, 1987, 1988; Keller *et al.,* 1991), and decreases in that activity will actually impede this defense. In addition, activation of the HPA axis during stress alters the production of cytokines and other mediators of inflammation, diminishing their action on several types of target cells.

Glucocorticoids play a major role in modulating the action of inflammatory and autoimmune responses and in influencing host susceptibility to disease by enhancing or inhibiting immune responses (McEwen *et al.*, 1997). However, current evidence indicates that adrenal steroids are not the only mediators of immune function and that their interaction with the autonomic nervous system, opiods and other neuropeptides, as well as protein hormones, also modulate immune function.

Corticotropin releasing factor acts to modulate immune function during stress (Webster *et al.*, 1998). Stress-induced secretion of hypothalamic CRF leads to secretion

of glucocorticoids and catecholamines, thus suppressing immune and inflammatory responses. But in addition, the immune system activates the HPA axis by cytokine/mediator (or inflammation mediated) stimulation of the CRF neuron and perhaps also of the pituitary zona fasciculata (Chrousos, 1995). This is a negative feedback cycle in which immune cell recognition of an infectious challenge activates the stress response by stimulating the secretion of CRF. During inflammatory stress, as the negative feedback cycle is activated, immune cell cytokines (predominantly the inflammatory related tumor necrosis factor-α, interleukin (IL-1, and IL-6) induce CRF secretion and activate the HPA axis as well as the sympathetic nervous system (Sapolsky *et al.*, 1987b; Webster *et al.*, 1998). Thus, in response to stimuli that normally function to enhance their production, leukocytes and lymphocytes are reduced. This negative feedback cycle is thought to prevent over-reaction of defense mechanisms and also to maintain the specificity of immune reactions (Munck *et al.*, 1984; Besedovsky *et al.*, 1986; Munck and Guyre, 1991). The presence of this negative feedback cycle suggests that the central action of CRF and glucocorticoids on the immune system may be to modulate immunologic function rather than simply to suppress it (McEwen *et al.*, 1997).

Numerous controlled studies of animals and humans have demonstrated the relative effects of various stressors on overall immune function (McEwen *et al.*, 1997) (Table 4). Psychological stressors have been demonstrated to cause immunosuppression in rodents (Keller *et al.*, 1983, 1991; Bohus and Koolhaas, 1991; Karp *et al.*, 1997). The repeated application of restraint and isolation stress compromised immune response to mitogens (Coppinger *et al.*, 1991; Minton *et al.*, 1992) and altered leukocyte differentiation antigens in lambs (Minton *et al.*, 1992). Similarly, a reduction in

lymphocyte proliferative responses was observed in stressed pigs exposed to mitogens (Brown-Borg *et al.*, 1993). Minton *et al.* (1995) found that lymphocyte proliferative functions were reduced in stressed lambs, but that increased levels of cortisol could not account for the reduced responses, implying that some factor other than cortisol may affect lymphocyte activity during stress.

In addition, chronic stress influences allergic, autoimmune/inflammatory, infectious, and neoplastic diseases (Webster *et al.*, 1997). One mechanism for this involves catecholamines, which are known to suppress immune function via adrenogenic receptor subtypes on immune cells (Madden *et al.*, 1995). Glucocorticoids and catecholamines produced during stress appear to favor the production of T helper (TH) 2 cells, which enhance humoral rather than cellular immunity, over that of TH 1 (Webster *et al.*, 1997).

Given the significant effects of stress on immune function, and the numerous studies indicating the immunosupressive effects of various stressors on mammals, it is plausible that fisheries-induced stress affects immune function in dolphins chased and captured by tuna purse-seine vessels. Although specific knowledge of immune function in spotted or spinner dolphins is lacking, research on the cetacean immune system has produced fundamental information on the identification of lymphocyte subpopulations and cell surface molecules (Romano *et al.*, 1992; Romano *et al.*, in press). In addition, studies of beluga whale lymphoid organ morphology and neural-immune interactions related to stress effects on immune systems have shown that the distribution of catecholamine-containing nerve fibers in the lymphoid compartments of these cetaceans are regional and specific (Romano *et al.*, 1994). This indicates that in response to stress,

norepinepherine and neuropeptides released from those fibers can alter immune response either by direct contact or via paracrine secretion (Romano, 1993; Romano *et al.*, 1993, 1994) .

One study investigated the effects of capture stress on the immune response of six beluga whales over a ten week period of captivity (St. Aubin *et al.*, 1990). The administration of a T cell dependent antigen elicited an immune response in these animals, indicating that the response was possible despite the stress imposed by capture. However, St. Aubin *et al.* (1990) cautioned that the results were preliminary (examining only one aspect of immune function) and that further work with larger sample sizes would be necessary to identify the effects of stress on the cetacean immune system. They noted that lymphocyte levels fluctuated over the period of captivity and that decreases in lymphocyte levels have been demonstrated in association with increased cortisol levels in stressed cetaceans (Medway *et al.* 1970; St. Aubin and Geraci, 1989).

3. Stress-Induced Changes in Reproductive Function

It is well known that stress can alter normal reproductive function in humans and animals (Moberg, 1976, 1987c, 1991; Coubrough, 1985; Rabin *et al.*, 1988; Rivier and Rivest, 1991; Dobson and Smith, 1995). In general, stress suppresses reproductive function in mammals. This has been suggested to be an adaptive response for the conservation of energy during times of hardship – whether caused by physiological or disease states, or environmental influences (Rabin *et al.*, 1988). Chronic stress, however, can affect reproductive success in wild populations. The adrenal stress response affects

reproductive efficiency and may have a central role in the self-regulation of mammalian populations (Christian *et al.*, 1965; Christian, 1971).

Management related stressors such as crowding, handling procedures, animal transport, intraspecies interactions, social status, psychological distress, noise, and physical trauma can have significant negative effects on the fertility of domestic animals (see Coubrough, 1985; Dobson and Smith, 1995). Specific examples of the negative effects of management stress include increased plasma cortisol levels and reduced ovulation in cows as the result of transportation stress (Edwards *et al.*, 1987). Reproductive problems in domestic animals subjected to transportation stress can persist for up to two months (Coubrough, 1985). Suppressive effects of stress disrupt estrus behavior in ewes (Ehnert and Moberg, 1991), as well as, estrus and ovulation in pigs (Hennessy and Williamson, 1983). Disrupted reproduction resulted from negative handling (prodding) and overcrowding in pigs (Hemsworth *et al.*, 1986a, b); but see Turner *et al.* (1998) who did not observe disruption of the reproductive cycle in pigs subjected to management related stress. Cows subjected to negative handling have had reproductive cycles disrupted (Stoebel and Moberg, 1982). Another frequently imposed management stressor, restraint, has been documented to have detrimental effects on reproduction in female Rhesus monkeys (Norman *et al.*, 1994).

a. *General Mechanisms*

There is a significant relationship between glucocorticoid hormones released by the HPA axis during stress, and the reproductive or hypothalamic pituitary gonadal (HPG) axis (Rivier and Rivest, 1991) (Figure 2). Activation of the HPA axis inhibits the

reproductive axis by inhibiting the luteinizing hormone releasing hormone (LHRH) neuron through the actions of CRF, β-endorphin, and glucocorticoids (Moberg, 1987c). In general, the duration of the stimulus, the interval between stimulations, and the intensity, or strength, of stressors are important factors in terms of the overall effects of these physiological mechanisms on reproduction.

Glucocorticoids affect the reproductive axis by inhibition of gonadotropin releasing hormone (GnRH) secretion, as well as by disrupting GnRH-induced LH release, and altering the effect of gonadotropins on sex steroid secretion (Moberg, 1987c; Rabin *et al.*, 1988; Rivier and Rivest, 1991). Glucocorticoids released during prolonged or acute stress inhibit pituitary gonadotropin secretion and gonadal function, and cause resistance of target tissues to sex steroids. For example, the gonadotropin induced release of LH is suppressed by elevated levels of glucocorticoids in many animals including rats (Baldwin, 1979; Rivier *et al.*, 1986; Du Ruisseau *et al.*, 1979), bulls (Welsh and Johnson, 1981), cows (Stoebel and Moberg, 1982; Eckternkamp, 1984), pigs (Pearce *et al.*, 1988), non-human primates (Moberg *et al.*, 1982; Sapolsky, 1985; O'Byrne *et al.*, 1988) and humans (Peyser *et al.*, 1973; Sowers *et al.*, 1979). In addition, prolactin can be elevated during stress, and may alter the LH surge in humans to cause infertility (Ben-David and Schenker, 1983).

It should also be noted that there have been cases where stress has increased LH release (Blake, 1975; Briski and Sylvester, 1988). This appears to be because stress activates hypothalamic noradrenergic neurons that exert complex stimulatory and inhibitory effects on GnRH release, and circulating levels of sex steroids act to modulate the LH response to stress (Rivier and Rivest, 1991). Brann and Mahesh (1991) suggested

that stress effects on inhibition or stimulation of gonadotropin secretion in females appears to be related to the length of exposure to a stressor and to background estrogen levels.

Corticotropin-releasing factor released during stress acts at the level of the brain to inhibit gonadotropin secretion, with β-endorphin opioids acting to modulate the interaction between CRF and gonadotropin release (Rivier and Vale, 1984; Petraglia *et al.*, 1986; Rivier *et al.*, 1986). For example, studies of amenorrheic anorexic humans have shown that decreases in gonadotropin secretion were associated with increased CRF and β-endorphins (Gold *et al.*, 1986; Gindoff and Ferin, 1987; Kaye *et al.*, 1987).

Corticotropin-releasing factor has an "extremely potent" suppressive effect on sexual behavior (receptivity) in the female rat (Sirinathsinghji *et al.*, 1983). Female rats administered exogenous CRF intracerebroventricularly not only exhibited suppressed receptivity, but also were aggressive and actively rejected male mounting attempts. This study demonstrates the direct effect of CRF on behavior and the role of CRF in affecting endocrine and behavioral responses to stress. Thus, in response to stress CRF may suppress reproduction by inhibition of female receptivity.

b. *Effects on Female Reproduction*

The stage from estrus to implantation appears to be most vulnerable to the effects of stress (Moberg, 1976). The delicate balance and timing of neuroendocrine events during the female reproductive cycle predispose her to potential interruption and reproductive failure resulting from stress. In particular, the follicular phase can be affected by stress and may be more susceptible to disruption than the luteal phase because

the neuroendocrine regulation of follicular development and ovulation is dependent upon interaction between the hypothalamus, pituitary, gonadotropins, and feedback activities of estradiol (Moberg, 1991).

In addition, stress in females can cause delayed puberty, lack of behavioral receptivity, irregular estrus, delayed ovulation, failure of ovulation or embryo implantation, spontaneous abortion, and increased infant mortality (Bachman and Kemmen, 1982; Sirinathsinghji *et al.*, 1983; Adams et al, 1985, Loucks *et al.*, 1989; Jurke *et al.*, 1997; also see Johnson *et al.*, 1992). Stress can also result in cystic ovarian degeneration. Matteri and Moberg (1982) noted that ACTH can cause cystic follicles in domestic livestock, and suggested that the development of ovarian cysts may be an additional result of insufficient LH release caused by high levels of ACTH during stress (see also Liptrap, 1970; Liptrap and McNally, 1976).

Lactation can also be altered as the result of stress. Prolactin levels, which are generally increased during lactation, affect milk production in mammals and can also be increased in response to stress (Collier *et al.*, 1984). In addition, mammary glucocorticoid receptors increase at lactogenesis, and growth hormone levels, also altered by stress, influence milk yield (Collier *et al.*, 1984). Lactation provides nutrition and passive immunity to the offspring (Guidry, 1985), and requires nearly three times as much energy from the female as does gestation (Millar, 1977). The energetic cost of lactation is nearly 80% of the total reproductive cost in many mammals (Oftedal, 1985). Milk energy output, milk yield, and milk composition all vary according to lactation stage and nutritional status. In response to chronic stress, energy demands upon the female can decrease milk yield and can even terminate lactation. If chase and capture

have more than temporary effects on the quantity or quality of milk produced by females with dependent calves, the calves may suffer nutritional deficiency with all of its ramifications.

C. *Effects on Male Reproduction*

Glucocorticoids produced during stressful events exert direct action on testis function by suppressing testosterone levels (Liptrap and Raeside, 1975; Pitzel *et al.*, 1979; Johnson *et al.*, 1982; Knight *et al.*, 1982, Cumming *et al.*, 1983). The effects of changes in testosterone level on male fertility are not known. Even under conditions where glucocorticoids have acted initially to increase testosterone levels in boars (Juniewicz and Johnson, 1981; Liptrap and Raeside, 1975) continued elevation of glucocorticoids appear to suppress testosterone release (Moberg, 1987c). Restraint stress significantly lowered testosterone levels in rats (Charpenet *et al.*, 1981). Stress induced by capture and immobilization lowered testosterone concentrations in the blood of free-ranging olive baboons (*Papio anubis*), was interpreted to imply that male reproduction can be inhibited by both psychogenic and neurogenic stressors (Sapolsky, 1985).

In summary, it is clear that both acute and chronic stress can have negative effects on reproduction in mammals. By implication, fisheries-induced stress may have multiple effects on reproduction in exploited populations of spotted and spinner dolphins. There is considerable information on reproduction in spotted dolphins (see Perrin and Hohn, 1994). Data on ovulation rate in spotted dolphins (estimated to be about 0.4 to 0.6 per year in this species) is partially based upon studies of individuals killed in the ETP fishery (Perrin *et al.*, 1976; Perrin and Reilly, 1984). Perrin *et al.* (1976) observed that

estimates of reproductive parameters for individuals from the ETP were different than those for individuals from the western Pacific, and concluded that the differences may have been due to activities of the tuna fishery. Reproduction is apparently seasonal in spotted dolphins in the ETP, with peak calving seasons in the spring and fall, and possibly in the summer (Perrin *et al.*, 1976). Length of lactation is about 1 to 2 years, and varies among populations. Changes, over time, in length of lactation within spotted dolphin populations may be related to fishing pressure (see Chivers, 1992; Perrin and Hohn, 1994).

Barlow (1985) compared reproductive rates in two populations in the ETP, one considered to be more exploited than the other, and observed a lower percentage of pregnant females in the exploited stock. This is contrary to what might be expected if density compensatory effects were operating (Barlow, 1985). The number of animals lactating and the number of immature animals were higher in the exploited population, which were also responses counter to what might be expected.

Reproductive patterns of spinner dolphins have been reviewed by Wells and Norris (1994) and Perrin and Gilpatrick (1994). Benirschke *et al.* (1980) examined the reproductive tracts of spinner dolphins killed in the ETP and suggested that ovulation is spontaneous in these animals. Ovulation rate for animals in the ETP has been estimated to be approximately one per year, and declines with age (Perrin *et al.*, 1977). Reproduction is seasonal in ETP spinner dolphins (Perrin *et al.*, 1977).

In comparing reproductive rates of two ETP spinner dolphin populations, Perrin and Henderson (1984) observed a lower percentage of pregnant females in the more heavily exploited eastern spinner stock than in the less exploited whitebelly spinner

stock. Once again, this observation is contrary to expected density-dependent changes for a population whose abundance is decreasing (Barlow, 1985).

Chivers and DeMaster (1994) analyzed potential biological indices for the northeastern and western/southern management units of spotted dolphin, and the eastern and whitebelly spinner dolphin. Compensatory responses were observed for the parameters examined indicating that responses to changing population abundance were occurring. However, not all changes in the parameters were consistent with the predictions for density dependent population responses. For eastern and whitebelly spinner dolphin populations, the proportions of pregnant females decreased over the time series analyzed. The authors suggested that time lag in responses or sampling biases may be responsible for some of the observed changes, but this does not exclude the possibility that fishery-induced stress may have been acting to decrease pregnancy rate.

The above information indicates that both the immediate effects of stressors to dolphins involved in the ETP fishery (activation of the HPA axis) and long-term effects could have adverse impacts on reproduction in these animals. The information also indicates that female reproduction may be particularly vulnerable to stressors.

4. Stress-Induced Changes in Growth

Chronic stress can inhibit growth in several ways. First, activation of the HPA axis suppresses growth hormone (GH) and inhibits the effects of insulin-like growth factors (IGF-I) on target tissues (Diegez *et al.*, 1988; Stratakis *et al.*, 1995). Second, CRF has been demonstrated to cause secretion of hypothalamic somatostatin, which inhibits

GH secretion (Ono *et al.*, 1984; Rivier and Vale, 1985). Also, glucocorticoids may act to make tissues resistant to growth factors.

In addition, physical contact between immature animals and caregivers is important for normal growth. Disruption of the infant-caregiver bond can affect growth (Schanberg and Kuhn, 1980; Schanberg and Field, 1988). A condition known as "psychosocial short stature" or "psychosocial dwarfism" in humans is caused by psychological abuse or stressful psychosocial environment (Johnson *et al.*, 1992; Stratikis *et al.*, 1995). The condition appears to be caused by disruption of social relationships (especially those involving the primary caregiver) during early development (Powell *et al.*, 1967). Consistent with the theory that the short stature results from psychosocial stress, deficient levels of GH and IGF-I have been documented to occur in patients with the condition (Albanese *et al.*, 1994).

The suppression of thyroid function also affects growth. Corticotropin releasing factor induced secretion of somatostatin suppresses secretion of the thyroid hormones, TRH and TSH. Also, glucocorticoids decrease the conversion of T4 to T3 during stress. Studies in rats have demonstrated that stress-induced decreases in TSH are influenced by age, with younger animals being more susceptible (Cizza *et al.*, 1995, 1996).

The effects of stress on growth may be a real concern in relation to fisheries interactions in the ETP if stressors such as maternal-young separation, separation from social group, novelty, and isolation have an impact on dolphin calves. Growth in young animals can also be affected secondarily if stress effects milk quality or yield in the mother or metabolism in the calf.

5. Stress-Induced Changes in Metabolism

Metabolism is affected by stress in mammals because activation of the HPA axis can increase metabolic rate and decrease nutrient absorption (see Elasser *et al.*, 1995). Mizrock (1995) reviewed what is known about alterations in carbohydrate metabolism during stress. In his general overview of stress and stress related diseases, Sapolsky (1998) emphasized the inefficiency of repeated mobilization of energy reserves in response to intermittent stressors. Chronic exposure to glucocorticoids, causing a depletion of energy reserves, can result in muscle wastage (Kaplan and Nagareda Schimizu, 1963).

Exposure to glucocorticoids also causes hepatic gluconeogenesis resulting in hyperglycemia, and can result in elevated triglyceride levels. A pseudodiabetic state can develop in tissues where glucose uptake is inhibited, and both lipid and protein catabolism can become pronounced. Ketosis, hyperlipemia, hyperaminoacidemia, and associated metabolic acidosis can develop as a result of these effects (Breazil, 1987). Other severe complications can arise from chronic exposure to elevated levels of glucocorticoid steroids. In humans and non-human primates, stress has been documented to cause insulin resistance and can lead to a state similar to that of a metabolic disorder known as "syndrome X" (Jayo *et al.*, 1993; Pasqualli *et al.*, 1993). The syndrome is characterized by obesity, insulin resistance, and increased occurrence of arteriolosclerosis.

The effects of fishery-induced stress on metabolism in spotted and spinner dolphins are entirely unknown. Based upon information from other mammals, it appears

possible that their metabolism could be affected by stress, but the potential impact on individual dolphins can not be assessed.

III. DISCUSSION

A. Potential Immediate Effects Of Stress

Capture and pursuit cause stress, as assessed by adrenocortical activity, to terrestrial mammals (Hattingh *et al.*, 1988, 1990; Harlow *et al.* 1992; Bateson and Bradshaw, 1997) and cetaceans (Thomson and Geraci, 1986; St. Aubin and Geraci., 1990; St. Aubin *et al.*, 1996). Capture stress in cetaceans elicits an adrenal response (elevated levels of cortisol and aldosterone) as well as changes in thyroid hormone balance, glucose, iron, lymphocytes and eosinophils (Thomson and Geraci, 1986; St. Aubin and Geraci., 1990, 1992; St. Aubin *et al.*, 1996).

Based on the literature from studies of stress in mammals, it is apparent that there will be differences in stress-related responses among species and individuals. Differences in temperament, age, sex, and health condition are all likely to influence the response of dolphins in the ETP to fisheries activities. Differences in experience with the fishery may also influence responses to stress among individual dolphins. Further, each encounter with the fishery will differ and this too will influence the potential response of an individual dolphin.

During chase and capture operations of the ETP tuna purse-seine fishery, dolphins encounter multiple stressors. In this review, specific stressors have been identified that could plausibly elicit an immediate physiological stress response of the hypothalamic-pituitary-adrenal axis. Chronic stress or repeated acute stress can have maladaptive

effects on immune responses, reproductive function and growth among others (Moberg 1991, 1987c; Rivier and Rivest, 1991; Chrousos and Gold, 1992; McEwen et al., 1997). Many of the types of stressors encountered by dolphins in the tuna purse-seine fishery have been shown to be pathogenic in other mammals under chronic or multiple acute influence.

One additional potentially significant aspect of search, chase and encirclement of dolphins in the ETP, is that the inter-species association between dolphins and tuna is disrupted. This may be important if there are nutritional advantages for the herds associated with tuna (see Au and Pitman, 1986). It appears that spotted and spinner dolphins may be largely nocturnal feeders (see Scott and Cattanach, 1998), but prey species in the ETP are patchily distributed and it is possible that dolphins aggregate with tuna, perhaps to maintain their proximity to prey species during the day, thus maximizing foraging opportunities at night.

It has also been observed that herd sizes of spotted and spinner dolphins appear to increase from night to daytime (Scott and Cattanach, 1998). The larger daytime aggregations have been suggested to provide more effective searching for prey that will migrate vertically at night and more opportunities for mating (Scott and Cattanach, 1998). As they react to search and chase by running from the helicopter and vessels, dolphins are displaced from the area where they were found. Habitat utilization, foraging efficiency, and social activities are all likely to be disrupted.

Many of the fishery-induced stressors are likely to have immediate physiological effects on individual dolphins. First, the perception of pursuit and lack of control are can elicit an HPA response in dolphins during their reaction to approach by vessels or

helicopters. Second, the potential for social separation and novelty of the conditions enforced by any given catch can also elicit an immediate HPA response in these dolphins. The stress of social separation may be particularly acute because these pelagic dolphins of the genus *Stenella* are herd animals. These dolphins exhibit strong social cohesiveness and structural integrity within herds. For example, Norris and Johnson (1994) emphasized that "a spinner dolphin never leaves the protective confines of its school." The instinct for herding in these animals appears to be strong and the benefits are presumably large. Although herd composition can be remarkably fluid, with fluctuating changes in group size and composition (Norris and Dohl, 1980a,b; Norris and Johnson, 1994; Scott and Cattanach, 1998), social relationships within these herds appear to be fundamental to individual survival. Because dolphins apparently have a relatively sophisticated communication system, herding affords the dolphins protection in the form of early warning about predators, organization against attack, and could allow conveying of information regarding prey or environmental change. Further, in his review of delphinid social ecology, Heimlich-Boran (1993) found that there appears to be a high degree of behavioral coordination among social groups in both spinner and spotted dolphins. By implication, there may be repeated interactions between cooperative groups, and this type of behavioral cohesiveness usually typifies kin-related groups (Heimlich-Boran, 1993). Given the probable importance of social relationships among dolphins and the benefits conferred upon individuals by herding, separation, when it does occur, is likely to elicit an adrenocortical response in these dolphins.

Another probable stimulant of immediate physiological stress response for dolphins encircled in purse-seine nets is the enforced disruption of spatial distribution

among dominant and subordinate animals confined within the nets. The central indication for this results from the work of Pryor and Kang Shallenberger (1991). In their observations of focal animals sampled from spotted dolphins in tuna purse-seine sets, they found that most occurrences of social aggression (threat displays or aggressive physical contact) could be interpreted as normal behavior and most incidences occurred between cow-calf pairs. Outside of focal animal observations, however, they documented many occurrences of "more extensive episodes" of social aggression. Most of these episodes were between adult males, but aggressive interactions between adult females, subadults, and one incident between a cow-calf pair were also observed. These are the types of interactions that have been demonstrated to elicit an HPA response in terrestrial mammals, and this type of stress associated with social rank has been shown to affect reproduction in primates (Sapolsky, 1985, 1987a; O'Byrne *et al.*, 1988; Creel et al., 1996).

In addition to eliciting an adrenocortical response, fisheries operations may have several other immediate physiological consequences to individual dolphins, including muscle damage and hyperthermia. For example, passive behavior of dolphins captured in tuna nets has been suggested to result from the effects of chase and capture on dolphin physiology (Coe and Stuntz; 1980; Sevenbergen and Myrick[3]). In particular, capture myopathy is suspected to occur in these dolphins (Stuntz and Shay, 1979). Some form of muscle damage or capture myopathy seems likely to occur in a portion of individual dolphins chased and captured in purse-seine nets, and, given the occurrence of capture

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3 Sevenbergen, K. L. and Myrick, A. C. Behavioral and physiological responses to stress in mammals: Criteria for evaluating dolphin behavior during chase and capture by purse seines in the eastern tropical Pacific. Unpublished manuscript cited with permission of K. L. Sevenbergen (23 November, 1998).

myopathy related mortality in terrestrial mammals (see Table 3a), it is plausible that post-capture mortality results from chase in some individuals. Death from capture myopathy could occur either as an endpoint of the condition itself or as the result of impaired ability to forage and avoid predation.

Cowan and Walker (1979) found no evidence of capture myopathy from observations of skeletal muscle obtained from dolphins killed in the ETP fishery. This may be evidence that muscle damage does not occur in dolphins as the result of chase and capture. Alternatively, based on information from terrestrial mammals, it seems possible that myopathic changes to skeletal muscle may occur gradually, during and post-capture, as the cumulative effect of systemic changes caused by the delayed-peracute syndrome. If available, data from examinations of newly captured or stranded cetaceans (representing a condition of severe stress) may provide insight to the cetacean stress response itself, and to the time course of resultant pathologies (Geraci and St. Aubin, 1977; Dierauf, 1990).

Capture myopathy-related injury to the heart, suffered in acute shock, can cause death. Turnbull and Cowan (1998) hypothesized that dolphins are particularly susceptible to stress cardiomyopathy. The authors found lesions typical of those attributed in other species to direct myocardial injury from catecholamines, or from coronary artery branch spasm induced by catecholamines or autonomic discharge ("contraction band necrosis" or CBN), in stranded dolphins (Turnbull and Cowan, 1998). In investigation of hearts from human drowning victims, CBN has been considered to be the cause of sudden death (Lunt and Rose, 1987). Given the conclusions of Cowan and Walker (1979), that stress cardiomyopathy occurred in dolphins killed in tuna purse-seine

nets, it is seems possible that CBN could lead to the death of animals captured in the ETP fishery.

One last immediate physiological response to stress must be noted here. Neurohormones secreted during stress can have an immediate effect impairing fertility in females (Moberg, 1991). The HPA response to stress could result in the disruption of neuroendocrine processes that govern the female reproductive cycle (Moberg, 1987c, Rivier and Rivest, 1991). Because this may be particularly true when females are in the follicular phase of reproduction, logic dictates that fishery exploitation coinciding with reproductive season in a spinner or spotted dolphin population could have a significant effect on the reproductive success of that population. It is also relevant that fishery-induced stress could suppress testosterone levels in dolphins, but the potential immediate effects, if any, on male fertility are not clear.

B. Potential Long-Term Effects Of Stress

The long-term effects of fishery-induced stress are difficult to detect. These are gradual effects occurring over time. These effects could include changes in immune status, as well as impaired reproduction. Romano (1993) suggested that stressors encountered by cetaceans in the wild (entanglement in fishing gear, social separation, contact with environmental pollutants such as oil or noise, and even extreme temperature changes) could compromise the immune system. She noted that immunologic defenses may be seriously compromised if pathogens such as viruses, bacteria, parasites, or toxins are present, and compounded with the effects of stress (Romano, 1993; see also Riley 1981, Welsh *et al.*, 1991). Although nothing is known about the immunocompetence of

dolphins involved in the tuna purse-seine fishery, it seems likely that compromises in immune function occur in response to fisheries-induced stress. If samples from dolphins killed in the fishery could be obtained, immunohistochemistry of spleen, thymus and lymph tissue (and possibly tonsils and additional lymphoid tissues) could be used to investigate the immune status of individuals given that background information on life history and health status of the animals are known. Suppression of natural killer cell activity due to stress (see Breazile, 1987, 1988; Keller *et al.,* 1991) has recently been observed in beluga whales using two independent methods (De Guise *et al.*, 1997), and may be another important indicator of immune system status in dolphins involved in the fishery.

It also seems likely that the reproduction of some proportion of female dolphins will be disrupted, either as a result of the HPA response to stress or through the development of pathologies resulting from chronic stress. Investigations of spotted and spinner dolphins have shown that high levels of fishery mortality were likely to have caused changes in reproductive parameters of exploited populations (see Perrin *et al.*, 1996, Barlow, 1985). One plausible explanation for the observations of a lower percentage of pregnant females in exploited stocks of spotted and spinner dolphins (Perrin and Henderson, 1984; Barlow, 1985) is that fishery-induced stress was disrupting reproduction in those heavily exploited stocks, so that reproductive parameters were unable to adjust to changes in population density.

Also with respect to reproduction, the energetic costs for a lactating female responding to stress may compromise lactation in several ways. Fisheries-induced stress could play a significant role in disrupting reproduction in dolphin populations because the

duration of lactation in mammals is influenced by external pressures and changes in lactation length can in turn alter reproductive rates in mammals (Sadleir, 1984).

C. Effects Of Stress On Immature Dolphins

Norris *et al.*, (1978) indicated that dolphins do become separated during chase and capture in the ETP, and it has been suggested that cow-calf separation is likely to occur (Barlow, 1985). It appears that young animals may be particularly vulnerable to the impacts of fisheries operations in the tuna purse-seine fishery. First, maternal separation in mammals elicits an HPA response that may not decrease over time even after repeated events (Levine, 1993). Second, novelty elicits a strong HPA response in mammals and may be particularly likely to affect young animals. In addition, compromises in quality and duration of lactation could affect growth in calves. Finally, activation of the HPA axis during stress can lead to impaired growth (Stratakis *et al.*, 1995).

D. Conclusion

Beyond the recognition that any one of these pathologies may have maladaptive impact (including mortality) on an individual, the potential effects of stressors must be relevant to the well-being of dolphins and the success of populations in the ETP.

It is difficult at present to characterize risk imposed on dolphin populations by the tuna fishery. This is partially because there is little available information on capture frequency, which is necessary to assess levels of exposure among individuals. Some animals may only be exposed to occasional acute stress induced by chase and capture, while other populations in areas of greater fishing effort are exposed to chase and capture

events. Understanding the exposure to each of these levels of stress can be important to determining the impact of stress on the population.

Similarly, although each of the pathologies described above is the likely product of fishery-induced stress on individual dolphins, it is difficult to obtain direct measurements of the effects of stress on animals killed in the fishery. To obtain this type of information, large sample sizes of specimens incidentally killed in tuna purse-seine nets must be collected. Further, it is important to have comparative specimens that have not experienced fisheries-induced stress, and are of the same species or a closely related species.

Clearly, a systematic approach will be necessary to test the effect of stress on dolphin populations in the ETP. Information on induced changes in age-specific rates of survival and reproduction will likely be needed to estimate the effects of risk factors to a population, but this is problematic because current data regarding population dynamics are not available for existing stocks of dolphins in the ETP. Such data have not been collected since the late 1980's

One potential approach to estimating population level effects of stress has been demonstrated as a type of risk assessment simulation model for integrating the potential effects of stress with other risk factors and population characteristics such as age structure and fecundity in salmon (Schreck, 1998). However, few studies have yet been completed that consider the effects on a population of long-term exposure to stress. It is not clear how applicable these methods will be to questions about fishery-related stress in ETP dolphin stocks. Investigations conducted by Schreck and colleagues have encompassed, among others, measures of stress effects of immunocompetence (Maule *et*

al., 1989), reproduction (Schreck *et al.*, 1991), and energetic response to handling stress (Davis and Schreck, 1997) in salmon. Inclusion of these types of stress effects in simulation modeling allows for estimates of generation time to population extinction, and could provide for observation of the relative effects of specific stressors on the population. It is unlikely, however, that sufficient samples could be obtained to provide the necessary vital rate information and direct evidence of stress in a given stock.

Although this review of existing literature regarding stress in mammals can not provide a quantitative answer to the question of whether the tuna fishery is causing stress to affected dolphin populations, the available information and evidence point to the likelihood that physiological stress is induced by fisheries activities. It is therefore plausible, that stress resulting from chase and capture in the ETP yellowfin tuna purse-seine fishery could have a population level effect on one or more dolphin stocks.

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Table 1. Various stages of fisheries operations that are potential stressors to the dolphins involved.

Procedure	Potential Stressor
Search - (helicopter, purse-seiner)	perceived threat/lack of control/disturbance disruption of tuna-dolphin association, habitat utilization, foraging and social activities, altered distribution
Chase - (helicopter, speedboats)	disruption of social unit (particularly cow-calf separation) forced exercise noise (helicopter, speedboats, explosives*, purse-seiner)
Capture - (encirclement by purse-seine net)	further disruption of social unit confinement social aggression proximity to predators in the net noise (machinery, purse-seiner, speedboats) offloaded diesel fuel in the vicinity of the net$^{\Sigma}$ net canopy, collapse$^{\Delta}$

* Explosives are currently illegal for use during fisheries procedures.

Σ Fisheries observers have noted that on occasion, diesel fuel is offloaded in the vicinity of the purse-seine net during the time when dolphins are detained.

Δ Net canopy and collapse are situations that do not typically occur when precaution is taken during a set. When they do occur, these conditions can lead to entrapment of dolphins below the surface of the water and can induce panic or stress.

Table 2. Some of the primary neuroendocrine substances involved in physiological responses to stress in mammals.

Substance	Origin	Effect
Corticotropin Releasing Factor	hypothalamic	activates pituitary-adrenal axis (principal regulator of ACTH and β-endorphin secretion) and sympathetic nervous system
Oxytocin	hypothalamic (neurohypohysis)	stimulates pituitary ACTH secretion
Vassopressin (nonapeptide hormone)	hypothalamic (neurohypohysis)	stimulates pituitary ACTH secretion enhances renal tubule water permeability, stimulates hepatic glyconeogenesis, enhances vasoconstriction
Adrenocorticotrophin	pituitary	controls secretion of glucocorticoids from the adrenal cortex
Beta-endorphins (peptide hormones)	pituitary (cosecreted with ACTH)	regulate T-cell dependent immunoglobulin production, lymphocyte proliferation, and natural killer cell activity, analgesic effects
Glucocorticoids	adrenal	permissive and regulatory effects on cardiovascular function, metabolism, muscle function, behavior, and immune system
Angiotensin II (plasma enzyme)	hepatic	stimulates pituitary ACTH secretion; stimulates adrenal production of aldosterone (enhances water and sodium reabsorbtion and potassium excretion by renal tubules); initiates thirst and has vasoconstrictor effects, stimulates synthesis and secretion of vasopressin
Catecholamines (epinephrine, norepinephrine)	adrenal	stimulate pituitary ACTH secretion

Table 3a. Some documented occurrences of capture myopathy in mammals and birds. The number of animals captured and the number of animals observed to have capture myopathy are given. General indications of the condition were assessed by gross observation, blood chemistry, gross pathology, and histological examination. Mortalities were generally latent and were only observed because progress of the animal was monitored subsequent to capture.

Species	Number (n) Captured/Injured	Type of Exertion	Indications	Mortality	Reference
CETACEANS					
Bottlenose dolphin, *Tursiops truncatus*	3/1	handling, transport	gross signs, blood chemistry	none	Colgrove, 1978
CARNIVORES					
Sea otters, *Enhydra lutris*	41/1	capture, chemical immobilization	blood chemistry	none	Williams and Pulley, 1983
UNGULATES					
White-tail deer, *Odocoileus virginianus*	1/1	unknown	gross pathology, histopathology	1	Hadlow, 1955
	1/1	unknown	gross signs, blood chemistry, gross pathology, histopathology	1	Wobeser *et al.*, 1976
	164/6	capture, chemical immobilization, handling	na	6	Conner et al.,1987

Species	Number (n) Captured/Injured	Type of Exertion	Indications	Mortality	Reference
	415/na	capture, handling	gross pathology, histopathology	23 in 26 days	Berringer *et al.*, 1996
Roe deer, *Capreolus capreolus*	17/7	capture, handling, restraint, transport	gross signs	7 in 7 days	Gibbs *et al.*, 1975
Duiker, *Cephalophus sylvicultor*	1/1	transport	gross signs, blood chemistry, gross pathology, histopathology	1 in 36 hours	Wallace *et al.*, 1987
Mountain goats, *Oreamnos americanus*	11/6	capture/handling	gross signs, blood chemistry, histopathology	3 in 3 to 26 days	Hebert and Cowan, 1971;
Bighorn sheep, *Ovis canadensis*	634/95	chase, capture, handling, chemical immobilization	gross signs, blood chemistry	12	Kock *et al.*, 1987a
Pronghorn antelope, *Antilocarpra americana*	594/39	chase, capture, handling, transport	gross signs, blood chemistry, gross pathology, histopathology	39 (During pursuit, upon capture, or 2- 13 days)	Chalmers and Barrett, 1977
Sable antelope, *Hippotragus niger*	2/2	immobilization (n=1)	gross signs, blood chemistry,	1 in 30 hrs,	Wallace *et al.*, 1987

Species	Number (n) Captured/Injured	Type of Exertion	Indications	Mortality	Reference
		handling/transport (n=2)	gross pathology, histopathology	1 euthanized	
Dama gazelle, *Gazella dama*	1/1	chemical immobilization, transport	gross signs, gross pathology, histopathology	1 in 6 days	Wallace *et al.*, 1987
Springbok, *Antidorcas marsupialis*	8/8	chase, capture, handling, transport	gross signs, blood chemistry	none	Gericke *et al.*, 1978
Simitar horned-oryx, *Oryx dammah*	1/1	chemical immobilization, transport	gross signs, gross pathology, histopathology	1 in 12 hours	Wallace *et al.*, 1987
Arabian oryx, *Oryx Leucoryx*	1/1	chemical immobilization, capture, transport	gross signs, blood chemistry	euthanized after 10 days	Vassert *et al.*, 1992
Zebra, *Equis burchelli*	6/6	chase, capture, handling	gross signs, blood chemistry, gross pathology	3	Harthoorn and Young, 1974
Moose, *Alces alces*	18/1	chase, chemical immobilization handling	gross signs, blood chemistry, gross pathology, histopathology	euthanized after 46 hours	Haigh *et al.*, 1977

Species	Number (n) Captured/Injured	Type of Exertion	Indications	Mortality	Reference
Elk, *Cervus canadensis*	123/3	capture, handling, transport	gross signs, blood chemistry, gross pathology, histopathology	3	Lewis *et al.*, 1977
Muntjac, *Muntiacus muntjak*	10/2	capture, handling, restraint, transport	gross signs	2 in 7 days	Gibbs *et al.*, 1975;
BIRDS					
Greater flamingoes, *Phoenicopters ruber roseus*	na	chase, capture, handling, restraint, transportation	gross signs, gross pathology, histopathology	na	Young, 1967
Lesser flamingoes, *P. minor*	na	chase, capture, handling, restraint, transportation	gross signs, gross pathology, histopathology	na	Young, 1967
Wild turkey, *Meleagris gallopavo*	79	capture, handling transport	gross pathology, histopathology	60 euthanized	Spraker *et al.*, 1987
Bustards, *Chlamydotis undulata*	18/5	capture, handling	gross pathology, histopathology	5	Bailey *et al.*, 1996

Table 3b. Information regarding chase for some documented occurrences of capture myopathy in mammals and birds. No information on speed or intensity of chase was available for the studies listed below.

Species	Duration of Chase	Mode of Pursuit	Type of Capture	Reference
Bighorn sheep, *Ovis canadensis*	na	helicopter	drive-net, chemical immobilization, net-gun	Kock *et al.*, 1987a
Pronghorn antelope, *Antilocarpra americana*	15 to 50 minutes (4 to 15 km distances)	ground vehicles, fixed-wing aircraft helicopter	drive-trap	Chalmers and Barrett, 1977
Springbok, *Antidorcas marsupialis*	15 to 20 minutes	ground vehicles, fixed-wing aircraft	drop nets	Gericke *et al.*, 1978
Zebra, *Equis burchelli*	na (2 km distance)	na*	"seizing by ears and tail"	Harthoorn and Young, 1974
Moose, *Alces alces*	2 ½ minutes	helicopter	chemical immobilization	Haigh *et al.*, 1977
Greater flamingoes, *Phoenicopters ruber roseus*	na	boat	net	Young, 1967
Lesser flamingoes, *P. minor*	na	boat	net	Young, 1967

* Chase by vehicle and helicopter is noted in the introduction of this paper, but no specific mode of chase was described in the materials and methods section.

Table 4. Some stress associated changes in the immune system of mammals. Modified from McEwen *et al.*, 1997.

Type of stressor	Effect	Subject	Reference
Restraint	involution of the thymus	rats	Selye, 1936
Isolation	decreased lymphocyte proliferation in response to antigenic stimulation	rats	Joasoo and McKenzie, 1976
Restraint and Isolation	altered lymphocyte function	lambs	Minton *et al.*, 1992
	reduced lymphocyte proliferative function	lambs	Minton *et al.*, 1995
Noise	leukopenia	rats	Monjan and Collector, 1977
	decreased lymphocyte proliferation in response to mitogen stimulation	mice	Monjan and Collector, 1977
	reduced lymphocyte cytotoxicity	mouse	Monjan and Collector, 1977
Crowding	decreased lymphocyte proliferation in response to antigenic stimulation	rats	Joasoo and McKenzie, 1976
Electric Shock	decreased interferon production	mouse	Jensen, 1968
	decreased natural killer cell activity	rat	Shavit *et al.*, 1984
	decreased Il-2 production	rat	
	decreased expression of Il-2 receptors	rat	Weiss *et al.*, 1989

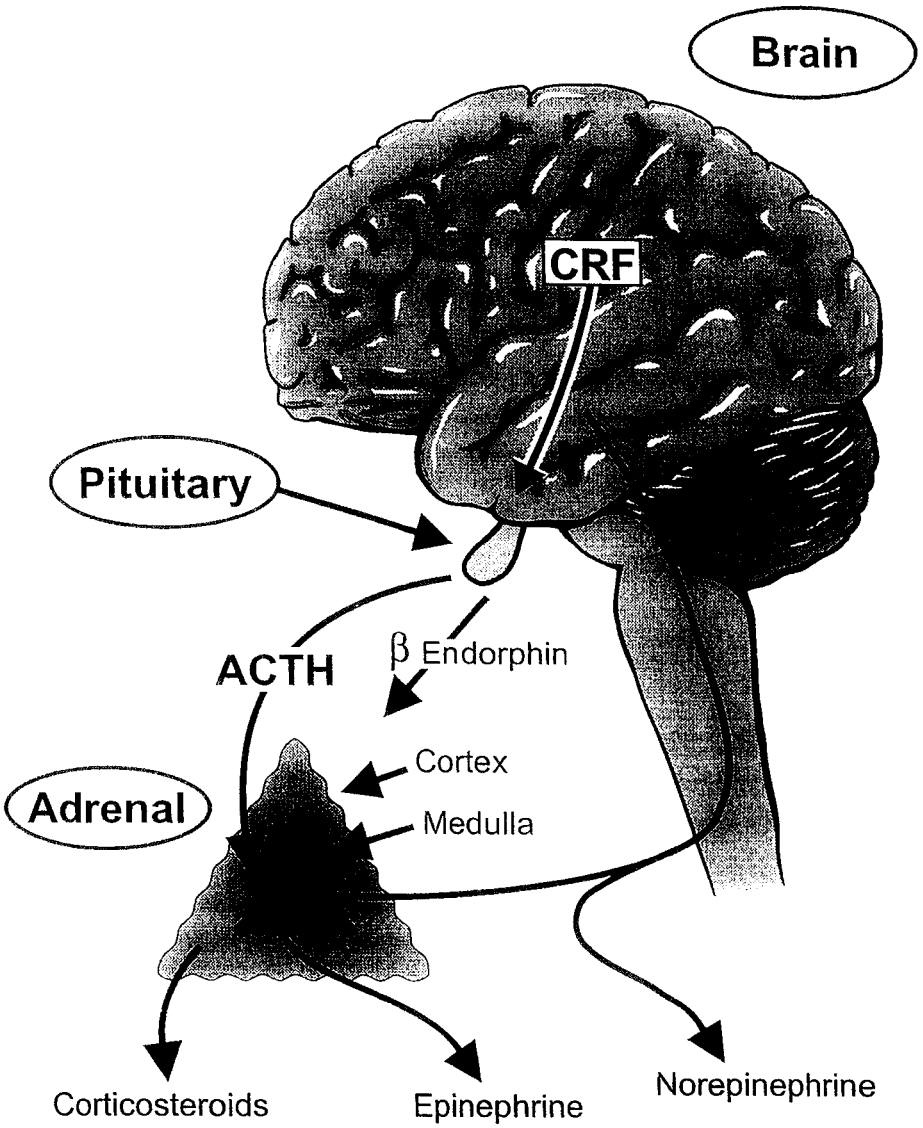

Figure 1. Schematic representation of the hypothalamic-pituitary-adrenal (HPA) axis. Corticotropin-releasing factor (CRF) facilitates physiological and behavioral responses to stressors. CRF activates the HPA axis and regulates secretion of adrenocorticotropin (ACTH) and β-endorphins. ACTH stimulates the adrenal cortex to produce corticosteroids. Epinephrine and norepinepherine are also produced in response to activation of the HPA axis.

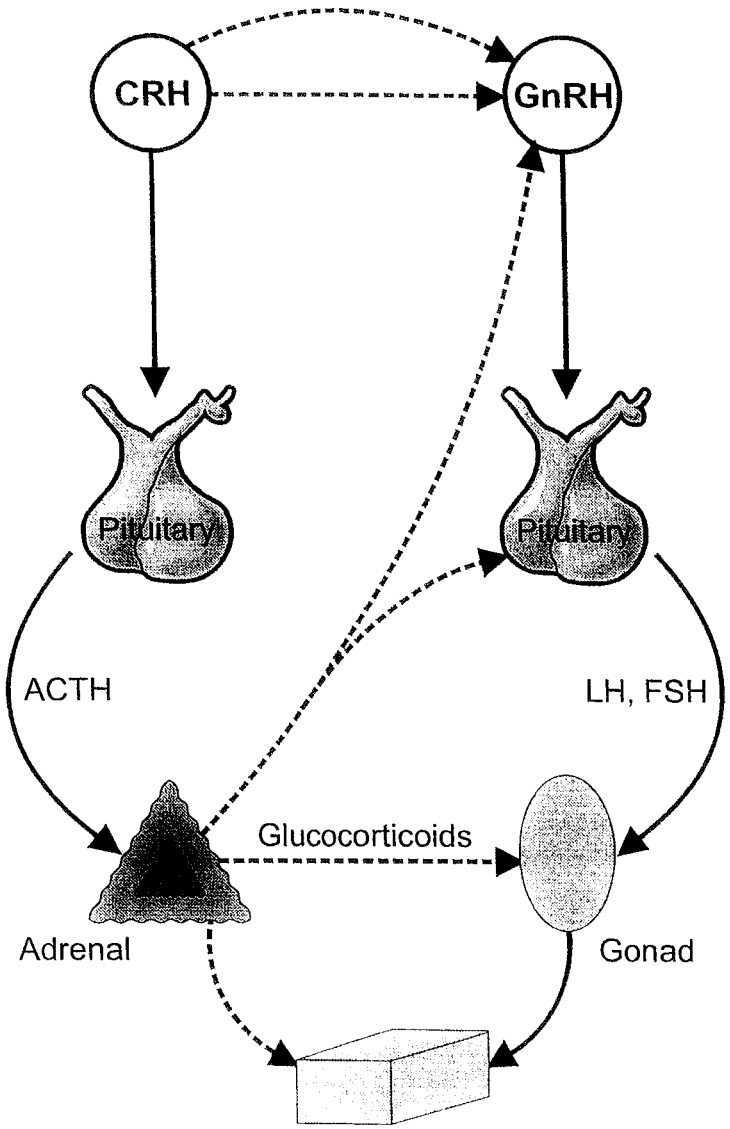

Figure 2. Schematic representation of the functional relationship between the hypothalamic-adrenal-pituitary (HPA) axis and the hypothalamic-pituitary-gonadal (HPG) axis. Corticotropin-releasing hormone (CRH) activates the HPA axis and gonadotropin-releasing hormone (GnRH) activates the HPG axis. The activity of GnRH secreting neurons is inhibited by CRH during stress. Glucocorticoids secreted by the adrenal gland suppress the activity of the HPG axis at all levels (hypothalamic, pituitary, gonads, and target tissues of sex steroids-luteinizing hormone (LH), follicle stimulating hormone (FSH)). Solid lines indicate stimulatory effects, broken lines indicate inhibitory effects. After Johnson *et al.* (1992).

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